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VETERINARY STUDENT ENROLLMENT FOR 1930-1931

It is safe to say that more eyes have been turned toward our veterinary colleges this fall than for many years. This is not surprising, in view of the fact that a great deal of attention has been directed to our veterinary institutions, during the past year, by a number of agencies interested in education in general and veterinary education in particular. The A. V. M. A. has been continuously interested in the subject of veterinary education for many years, but more recently such agencies as the Bureau of Education of our federal Department of the Interior, and the Rockefeller Foundation have seen fit to carry on some investigations in the form of surveys of the field of veterinary education in this country.

A question which has been debated, upon numerous occasions during the past ten years, has been the supply and demand of graduate veterinarians. The two questions which have been uppermost in the minds of those interested have been: Is there a surplus of veterinarians or is there a shortage of veterinarians, either now or impending? These questions seem to have been answered rather definitely by the report of the A. V. M. A. Committee on Education, made at the Los Angeles convention, and published in the October issue of the JOURNAL. That report very clearly shows that it will be necessary for our veterinary colleges

to graduate approximately twice as many veterinarians, in the future, as have been turned out during the past ten-year period, if we are to maintain our present numerical strength of approximately 10,000 veterinarians. Where our colleges have been graduating about 150 men each year, it appears necessary, from the figures submitted by the Committee, for this number to be doubled approximately, in order to fill up the ranks of the profession depleted by death and other causes.

During recent years, there has been a slow but steady increase in the number of freshman veterinary students enrolled each fall. There has not yet been time for this increase in freshman students to show itself materially in the number of veterinarians graduated, although the number of graduates has increased during the past three years, the low point having been reached in 1927, when all of the veterinary colleges in the United States and Canada graduated only 118 men. The number this year was 170.

Following a custom established in 1923, we are again presenting our annual report on veterinary student registration in the thirteen veterinary colleges located in the United States and Canada. It will be noted that the total number of veterinary students enrolled this year is 1246, compared with 1084 last year, or a net increase of 162 students. All of the colleges, with the single exception of Washington, report an increase in veterinary student enrollment this year. Washington shows a decrease of 16 from last year. Some of the increases are small, while Iowa State College shows the largest gain with 46 more students enrolled this year than last. Incidentally, it might be mentioned that Iowa has the largest number of students of any of the veterinary colleges, by a very considerable margin.

Turning to the freshman enrollment, we find that the thirteen freshman classes have a total registration of 429 students, as compared with 348 in 1929. This is a gain of almost 25 per cent. The gain in freshman students, however, has not been uniform throughout the list of colleges. Alabama, Cornell, Michigan, Iowa, Ohio, Ontario and Montreal show larger freshman classes than last year. Texas and Georgia report the same number as one year ago. The four other colleges have fewer freshmen this year than last. Iowa again leads the other colleges with an enrollment of 92 first-year students.

It is rather significant to note that although there were 348 freshmen enrolled last year, there are only 282 sophomores in the thirteen colleges this year. This is a loss of 66 students, and from

information that is available we are in a position to state that this decrease is, to a considerable extent, the result of several of the colleges dismissing students who were found to be incapable of continuing their courses. This is a good sign. What the veterinary profession needs, as much as anything else, is quality and not quantity.

With 20 freshman students enrolled at Alabama, Dean Cary reports the largest freshman class in the history of the institution. It is interesting to note that Dean Glover reports 29 students in each of the freshman, sophomore and junior classes at Colorado. The freshman class at Cornell this year is unusually large, with 58 first-year students reported by Dean Fish. Montreal reports no senior students this year. Dean Brumley reports 48 freshmen at Ohio. This is not a record, however, as there were 49 freshmen registered the fall of 1927.

With 215 senior students enrolled in the thirteen colleges, this fall, there is the prospect of having in the neighborhood of 200 graduates in 1931. This will be the largest number since 1923, in which year the total number of graduates was 251. It is rather unusual that the report this year should show 3 more juniors than sophomores. Ordinarily the sophomore students slightly outnumber the juniors. This is particularly true when the enrollment from year to year is on the upgrade.

Six of the colleges report a total of 24 graduate students. This is a healthy sign. However, the number should be materially

Veterinary student enrollment for the college year 1930-31

	Fr.	Soph.	Jun.	Sen.	Spec.	Grad.	Total	1929-30	Change
Alabama P. I.	20	8	10	4	0	0	42	34	+ 8
Colorado A. C.	29	29	29	15	1	0	103	98	+ 5
Cornell Univ.	58	28	40	34	0	3	163	135	+28
Georgia S. C.	13	12	14	8	0	0	47	41	+ 6
Iowa State Col.	92	53	42	40	0	6	233	187	+46
Kansas S. A. C.	41	43	21	15	4	2	126	118	+ 8
Michigan S. C.	14	8	11	8	5	6	52	42	+10
Montréal, Univ. of.	14	8	3	0	0	0	25	12	+13
Ohio State U.	48	28	36	31	0	6	149	124	+25
Ontario V. C.	36	21	23	19	0	0	99	81	+18
Penna., Univ. of.	40	27	25	19	1	1	113	107	+ 6
Texas A. & M. C.	7	5	7	6	0	0	25	20	+ 5
Washington, S. C. of.	17	12	24	16	0	0	69	85	—16
Totals	429	282	285	215	11	24	1246	1084	+162

increased. Our colleges should lend more encouragement to graduate veterinarians who desire to supplement the regular course with graduate work. At the present time graduate studies provide the only alternative for lengthening the regular four-year course. In most institutions the regular curriculum is so crowded that there is practically no opportunity for regularly enrolled students to take additional work.

DID YOU SUBSCRIBE?

A well-known dairy publication recently solicited subscriptions from members of the veterinary profession. A sample copy of the publication was sent to each veterinarian solicited. Here is what an examination of this particular issue disclosed:

A leading article dealing with fake cures for abortion disease of cattle; a display advertisement of "Herd Tonik," indicated in failure to breed, retention of the afterbirth, cow pox, garget and shortage of milk, and sold by the Dr. David Roberts Veterinary Company, of Waukesha, Wisconsin, with a photograph of dear old Doctor Roberts himself; a smaller advertisement of Lowell's Cow Cleaning Prescription (it *releaves* garget and is a *preventative* of milk fever); an advertisement of the Gizzard Capsules (for *seca* worms and others), marketed by the Geo. H. Lee Co., of Omaha, Neb.; a "veterinary" column, in which free advice is given on animal diseases, and—well, what more do you want?

Consistency, thou art a jewel.

IS A CHANGE IN ORDER?

Section 1, of Article 10, of the By-laws, in connection with Executive Board elections, provides as follows:

> In case of a tie vote, the President shall call a meeting of the members from the Executive Board district, in attendance at the annual meeting, and these members shall ballot for a choice between the tied nominees only.

The question arises as to whether or not this provision is a wise one. During the present year, elections were held in five Executive Board districts. In no case did an election result in a tie, although the previous year one vote decided an election in another Executive Board district. The point which we wish to bring out is that the provision of the By-law above quoted may place entirely too much responsibility in the hands of a very small number of members. If there had been a tie, this year, in District 9, the election would have been decided (?) by the

two members who were in attendance at the Los Angeles convention.

If there had been no choice in District 8, the election would have been in the hands of the seventeen members from District 8 who attended the convention this year. If there had been a tie in District 5, there would have been just eleven members at the convention, from this District, to decide the election, and if there had been no decision in District 10, the responsibility for selecting the Executive Board member for this District would have been in the hands of five members. Although no elections were held this year in District 2, 3 and 4, nevertheless very much the same situation would have presented itself by reason of the fact that there were just three members present from District 2, seven from District 3, and eight from District 4.

Of course, the meeting this year was unusual, in that it was held in a city located a considerable distance from the center of our membership. Nevertheless, the By-law above quoted would have prevailed if there had been any necessity for falling back on its provision. For the reasons above given, we question the soundness of the principle embodied in this particular section of the By-laws.

IT MAY WORK BOTH WAYS

A recent outbreak of hog cholera in Wisconsin was traced to the improper use of anti-hog cholera serum and virus by a registered non-graduate practitioner in Pierce County. Investigation disclosed that the practitioner had used inadequate doses of serum, with the natural result of producing hog cholera rather than preventing it. As a result of this incident the authorities have made a new ruling, which requires that all non-graduate veterinarians must take special training in the administration of anti-hog cholera serum and virus, before they can use these products.

The situation was given wide publicity throughout Wisconsin and the news release from the State Department of Agriculture and Markets was published in quite a number of local newspapers throughout the State. It was interesting to note the various captions placed over the same article in different papers. The Milwaukee *Sentinel* headed the article as follows, "Skill Needed in Vaccinating for Cholera," while the Woodville *Leader* ran the article under the following heading, "Cholora Outbreak in Pierce Co. Laid to Veterinarians."

We have frequently made the statement that publicity for the veterinary profession may be a two-edged sword. Here is an excellent example of just what may happen.

A PUBLIC BENEFACTOR

One of the best tributes to the veterinarian to come to our notice recently was in the form of an editorial in the September issue of *Successful Farming*, under the caption, "Veterinarian a Public Benefactor." We thought so highly of this editorial that it is being reproduced in full. It speaks for itself. Here it is:

> Most of us can remember when the community veterinarian was known as nothing more or less than a "horse doctor." In fact even as little as a quarter of a century ago horses were the only animals on many farms that were worth spending a veterinarian's fee to try to save.
>
> The pathetic part of it was that most of the veterinarians deserved no better recognition that that commonly given them. But improved live stock with a far greater value than formerly are forcing a change. Even the poultry flock today on most farms is worth paying a good veterinary fee to save if something goes wrong. But the veterinarian must be much more than the "quack" horse doctor of former years if he is to meet that need.
>
> As our land is taken up and becomes older and live stock population becomes more dense, disease and parasitic problems are multiplying rapidly. They will continue to increase as time goes on. The veterinarian who is capable of diagnosing and controlling disease and parasitic problems of the different kinds of live stock in his community today is of vast economic importance to that community. Many of us do not yet realize his importance because much of the damage done by disease and parasites is of such an unseen nature that it does not impress us. But as time goes on these losses will become more and more apparent and the good veterinarian will occupy a constantly higher place in his community.
>
> Only in recent years have we connected the veterinarian with human health. But because so many animal diseases are communicable to humans and because so many animal products are carriers of disease we are coming to realize that the veterinarian may be of real importance in controlling human disease. Only recently a representative of the U. S. Public Health Service made the statement that he would like to see a competent veterinarian represented on each board of health.
>
> Note that we are stressing competent, well informed and fair minded veterinarians. We already have lots of them and their number is increasing. But there are still entirely too many of the other kind. And our facilities in the way of high class, well equipped veterinary medical schools is entirely inadequate. One of the greatest needs of the live stock industry today is more good high class veterinarians. That need is increasing. It is highly important to the future of our American live stock, dairy and poultry industries that we properly support our good veterinary schools so that they in turn can help us eliminate our losses.

The above editorial contains considerable in the way of food for thought, not only for members of the veterinary profession but for those who control the purse-strings in those states maintaining veterinary colleges.

APPLICATIONS FOR MEMBERSHIP

(See July, 1930, JOURNAL)

FIRST LISTING

BOXMEYER, ROY EDMUND — 3614 Wyoming, Kansas City, Mo.
D. V. M., Kansas City Veterinary College, 1913
Vouchers: G. G. Graham and C. E. Salsbery.

CAIRY, FOREST WARD — Stock Yards Station, Sioux City, Iowa
D. V. M., Iowa State College, 1913
Vouchers: Louis D. Mersch and N. L. Nelson.

EMMEL, MARK WIRTH — Ala. Poly. Inst., Auburn, Ala.
D. V. M., Iowa State College, 1919
M. S., Michigan State College, 1930
Vouchers: C. A. Cary and H. Preston Hoskins.

HIRSCHER, HENRY L. — 4351 Broadway, New York, N. Y.
D. V. S., New York University, 1914
Vouchers: Sol Shapera and R. S. MacKellar.

HOLYCROSS, V. C., CAPT. FOREST LEE — Vet. Sta. Hospital, Fort Riley, Kans.
D. V. M., Ohio State University, 1914
Vouchers: William H. Dean and John A. McKinnon.

JOHNSON, SAMUEL RAY — 800 Woodswether Road, Kansas City, Mo.
D. V. M., Kansas State Agricultural College, 1930
Vouchers: Ashe Lockhart and G. G. Graham.

KERR, ALGERNON HUBBARD — Dudley, Route 2, N. C.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: W. C. Dendinger and Wm. Moore.

MINER, V. C., CAPT. JOHN W. — Fort Leavenworth, Kans.
D. V. M., Iowa State College, 1917
Vouchers: C. E. Cook and R. C. Musser.

PEDERSEN, HANS — 2600 Benton Blvd., Kansas City, Mo.
D. V. M., Royal Veterinary College, 1927
Vouchers: G. G. Graham and C. E. Salsbery.

RICHARDS, EARL DEAN — 1013 S. River St., R. 2, Austin, Minn.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: E. B. Carter and D. D. Tierney.

SHORE, CHARLES BRUCE — Oak Lane, Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1917
Vouchers: C. J. Marshall and Malcolm J. Harkins.

SULLIVAN, CORNELIUS FRANCIS — 74 Harbor View St., Dorchester, Mass.
V. M. D., University of Pennsylvania, 1930
Vouchers: D. S. Shannon and A. S. Cleaves.

SWANSON, LEONARD ERWIN — Apt. 5, 13th Ave., Columbus, Ohio
D. V. M., Ohio State University, 1927
Vouchers: Walter R. Krill and W. F. Guard.

WILLIAMSON, WALLACE LEROY — 401 W. Winona St., Austin, Minn.
D. V. M., Ohio State University, 1915
Vouchers: E. B. Carter and D. D. Tierney.

Applications Pending

SECOND LISTING

Anderson, Alexander R., Jr., 5956 Van Nuys Blvd., Van Nuys, Calif.
Baldwin, Frank Alfred, 1280 Grand Ave., Saint Paul, Minn.
Bickmore, Joseph Lewis, 647 Vine St., Sebastopol, Calif.
Brown, Allan K., Capitola, Calif.
Bussong, Harry L., Belton, Mo.

Coberly, James E., Box 694, Mesa, Ariz.
Collins, Horace R., Box 532, El Paso, Texas.
Colton, Cyril H., Box 101, Pomona, Calif.
Craig, Walter S., 1238 S. 3rd, Montrose, Colo.
Currey, John Raymond, 3622 Georgia Ave. N. W., Washington, D. C.
Darst, Lloyd Marion, Princeton, Ill.
Dibbern, Clarence John, 1016 Harriman Ave., Los Angeles, Calif.
Diller, Claude R., 7970 Santa Monica Blvd., West Hollywood, Calif.
Dungan, John Thomas, Box 282, Glendale, Ariz.
Gardner, Hyman W., 105-06 Metropolitan Ave., Forest Hills, L. I., N. Y.
Goodwin, Charles Edgar, Box 630, Picton, Ont.
Hendricks, Walstein Hyer, 648 Lake St., Salt Lake City, Utah.
Houston, Cecil, 523 Superior St., Milwaukee, Wis.
LaGrange, DeWitt, c/o Dr. J. B. Reidy, Box 327, Harrisburg, Pa.
Macintosh, Robert Duncan, c/o Tela Railroad Co., Tela, Honduras, C. A.
Marney, Floyd L., 2081 E. Randolph St., Huntington Park, Calif.
Pattison, Marvin L., 138 Lemoore Ave., Lemoore, Calif.
Pistor, William Jacob, 415 E. 7th St., Portland, Ore.
Scott, Richard M., 612 N. Western Ave., Los Angeles, Calif.
Sexton, William F., 931 N. LaBrea Ave., Hollywood, Calif.
Shepherd, Harry E., 6371 Cedar St., Huntington Park, Calif.
Stauffer, Jay Brown, Box 403, Harrisburg, Pa.
Tipton, Thos. B., Hume, Mo.
Watkins, Frederick Walter, Box 95, Santa Susana, Calif.
Wilkins, Harold L., Box 426, Anaheim, Calif.
Worrell, George W., Grenada, Calif.
Young, Elmer W., Fort Lewis, Wash.

The amount which should accompany an application filed this month is $5.83, which covers membership fee and dues to January 1, 1931, including subscription to the JOURNAL. It is suggested that applications filed this month be accompanied by remittance for $10.83, the additional $5.00 being for the 1931 dues.

COMING VETERINARY MEETINGS

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. November 5, 1930. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. November 5, 1930. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

Purdue University, Veterinary Short Course. Purdue University, Lafayette, Ind. November 5-7, 1930. Dr. R. A. Craig, Department of Veterinary Science, Purdue University, Lafayette, Ind.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. November 11, 1930. Dr. J. B. Jaffray, Secretary, 2956 Washington Blvd., Chicago, Ill.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. November 11, 1930. Dr. H. J. Hearrington, Secretary, 1130 Franklin Ave., Lexington, Mo.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. November 12, 1930. Dr. H. Preston Hoskins, Secretary, 537 Book Bldg., Detroit, Mich.

Tulsa County Veterinary Association. Tulsa, Okla. November 13, 1930. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Florida State Veterinary Medical Association. West Palm Beach, Fla. November 17-18, 1930. Dr. J. V. Knapp, Secretary, Box 445, Tallahassee, Fla.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. November 19, 1930. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Missouri Valley Veterinary Association. Aladdin Hotel, Kansas City, Mo. November 19-21, 1930. Dr. E. R. Steel, Secretary, 8043 Wornall Road, Kansas City, Mo.

Michigan-Ohio Veterinary Medical Association. Adrian, Mich. November 20, 1930. Dr. E. C. W. Schubel, Secretary, Blissfield, Mich.

Massachusetts Veterinary Association. Boston, Mass. November 24-25, 1930. Dr. Harry W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Keystone Veterinary Medical Association. Philadelphia, Pa. November 26, 1930. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

U. S. Live Stock Sanitary Association. LaSalle Hotel, Chicago, Ill. December 3-5, 1930. Dr. O. E. Dyson, Secretary, 45 Live Stock Exch. Bldg., Wichita, Kans.

Nebraska State Veterinary Medical Association. Lincoln Hotel, Lincoln, Nebr. December 9-10, 1930. Dr. Bernard Witt, Secretary, Scribner, Nebr.

American Association for the Advancement of Science. Cleveland, Ohio. December 29, 1930-January 3, 1931. Dr. Burton E. Livingston, Secretary, Smithsonian Institution Bldg., Washington, D. C.

STATE BOARD EXAMINATION

Nebraska State Board of Veterinary Examiners. Capitol Bldg., Lincoln, Nebr. November 12-13, 1930. Dr. F. R. Woodring, Secretary, Capitol Bldg., Lincoln, Nebr.

THE MUSEUM OF PATHOLOGY: A NEGLECTED INSTITUTION*

By WILLIAM H. FELDMAN, *Rochester, Minnesota*

Division of Experimental Surgery and Pathology,

The Mayo Foundation

For the last two years I have attempted to compile data pertaining to the existing collections in museums of comparative pathology. This effort has been made for the International Association of Medical Museums, which society is interested in compiling a comprehensive index of the medical museums of the world. A study of the information obtained so far, together with impressions formed after visiting several of our veterinary colleges, leads me to believe that the pathologic museums in many of the institutions which teach veterinary medicine have been relegated to a position of minor importance in the scheme of medical pedagogy. Instead of being regarded as a necessary part of the teaching equipment, which needs constant and sympathetic encouragement from all members of the faculty, the pathologic museum is often regarded with more or less indifference by all except the pathologist, who usually is occupied with routine duties so as to preclude the giving of sufficient time to the museum to enable it to become more than a nondescript collection of biologic curiosities.

The importance of a proper museum collection in the teaching of the medical sciences cannot be over-emphasized. At this time, when the leaders in the field of veterinary education are examining the existing methods of teaching as well as the course of study, some consideration might well be given to the possibility of broadening the student's knowledge, not only of fundamental subjects such as anatomy and general pathology but of the clinical branches, by making full use of museum facilities available. When the real pedagogic value of a well-arranged, although small, collection is appreciated, this part of the teaching equipment will then assume its rightful place as an institution of genuine academic indispensability. In fact, "the ideal pathologic museum must be a teaching institution and must be primarily organized for teaching purposes."[1]

*Presented at the sixty-seventh annual meeting of the American Veterinary Medical Association, Los Angeles, Calif., August 26-29, 1930.

The medical schools have long understood the necessity of maintaining properly conducted pathologic museums in the furtherance of medical education. Several years ago, in commenting on the absence of proper equipment in some of the medical colleges, Flexner[2] said: "The conclusive evidence of lack of educational conscience or pride is the general absence of a decent museum." More recently, Ewing[3] wrote:

> The value of a comprehensive pathological museum for teaching students, clinicians and pathologists, and for research in the etiology and pathogenesis of disease, is probably underestimated even by the most confirmed pathological anatomists.

The benefits that accrue to the student fortunate enough to attend an institution in which full use is made of the museum in the form of objective teaching are many, and compensate fully for the time and money expended in the creation of such a collection. Abbott[1] has set forth the value that may be derived by the student in the acquisition of knowledge by this means:

> His native powers of observation, judgment, and induction are at once elicited; he obtains a new understanding of what he has read and seen elsewhere. . . . this gain of knowledge or insight into disease, is acquired without a strain on his memory but rather with the mental relief which comes from seeing in their simplicity conditions, the abstract ideas of which it has required often a powerful effort to carry in his mind. The organized museum is to general pathology what the autopsy room is to medicine, what dissection is to anatomy, what—to go further afield—traveling to see new countries is to the study of geography.

In attempting to analyze the reasons for the failure of the pathologic museum to achieve its proper position in the teaching equipment of many of our veterinary schools, several pertinent factors come to mind. The teaching staffs often lack appreciation of the pedagogic value of a well-arranged and ever-enlarging museum, although nothing can equal it as a means of imparting knowledge pertaining to disease with all of its complex manifestations. Ideas gained by visual impressions seldom are tainted by erroneous interpretations which not infrequently are imparted by the printed page or even the spoken word. Vague notions acquired in the lecture-room or indefinite impressions obtained in the laboratory are quickly and permanently expelled by a few minutes spent in the museum in visual contact with a typical specimen depicting the particular point in question. It is my opinion that if those responsible fully understood the benefits to be derived from a museum for teaching purposes, such a plea as I am attempting to make would not be necessary.

In some instances the lack of suitable space for the accommodation of a museum may constitute a plausible reason why the

collection has not been expanded in a manner comparable with the growth of other departments. On one hand, it is proper, of course, that lectures and laboratory classes be given preference over museum collections, but on the other hand, it is possible to utilize various walls, corners, and other odd nooks in such a fashion as to provide at least temporary space for a collection which would be of inestimable value to the classes in general pathology, inspection of meat, and parasitology. If the specimens are chosen discreetly and the collection is properly preserved and displayed, with the proper guidance it should be accepted by teachers and students alike as a teaching necessity. With the inevitable growth of the collection, institutional pride, with administrative wisdom, usually will provide space in which the museum can be suitably and permanently housed. If proper space for a museum is really wanted and necessary, it would at least seem expedient to create a creditable collection first, since experience indicates that once the collection assumes commendable proportions it eventually finds its way into proper accommodations. The argument that a museum cannot be developed because of lack of suitable room is not entirely valid. Certainly the absence of a specially designated room does not constitute an obstacle of any significance in the furtherance of the museum idea.

Lack of acceptable material also may be offered as a reason why a given collection maintains such meagre proportions. Unless the museum is constantly acquiring new material, the specimens gradually decrease in numbers due to deterioration caused by various factors. A neglected museum is a diminishing one. If the museum is worthy of the name, new specimens must be added constantly. The addition of new material not only enhances the intrinsic value of the collection but stimulates to a marked degree the interest of those who use it.

New material is not difficult to secure from such obvious sources as practitioners, veterinary hospitals, and abattoirs that are supervised by the federal government. The last-named are indeed fruitful sources of pathologic material of the common meat-producing animals, and it has been my experience that the majority of veterinarians in the federal meat inspection service will extend their fullest coöperation, as far as their official duties will permit, in the collection of any particular material which may be desired. Proper authorization must be secured before specimens can be obtained from this source, but this usually

entails no particular difficulty. The large amount of pathologic material encountered every week in the larger packing-centers constitutes a practically inexhaustible supply of splendid specimens for museum purposes. This material can be secured perfectly fresh, and consequently is capable of being prepared in a manner which will insure proper fixation and the preservation of natural colors. It has the advantage over much of the usual material obtained from necropsies conducted in veterinary hospitals or secured from those who perform a necropsy on the farm. Specimens obtained from the occasional necropsy performed on the farm or in the veterinary hospital often are impossible to preserve for display purposes on account of advanced postmortem changes.

While meat inspectors encounter a great variety of pathologic conditions acceptable for museum collections, the relative frequency of tuberculous lesions in the various species makes it possible for every museum to have a first-class collection of material depicting the usual as well as the unusual manifestations of this important disease. If such a collection were possessed by the respective veterinary colleges, it would enable the students who enter the meat inspection service to obtain a first-hand knowledge of this common disease which usually is obtained only after entering on their official duties.

Among other important diseases, of which a complete group display might be arranged from material obtained from abattoirs, are hog cholera, the so-called lymphoid hyperplasias (the leukemias), parasitic infestations, actinomycosis, and neoplasia. Some of our museums already contain excellent collections illustrating these conditions, but unfortunately there are many which do not. Too often a rather common disease is represented by a single specimen and the student, if he sees it at all, may as a consequence infer that the disease in question is of relatively little significance. The comparative importance of the respective diseases can well be emphasized by the relative number of specimens displayed.

The abundance of excellent pathologic material which can usually be obtained for the asking should stimulate all of us interested in comparative pathology to aid in securing for our teaching institutions a comprehensive collection comparable at least with those maintained by the best medical schools.

Another hindrance to the fullest development of the pathologic museum is the shortage of funds for the purchase of proper

museum equipment. This is a serious problem, the solving of which necessitates considerable thought. However, less money might be expended for elaborate and expensive display cases in order to provide sufficient and appropriate containers for specimens. Ordinary tables, with pyramid-like tiers, and wall-shelves can be utilized in such a manner as to make the individual specimens of the collection even more accessible than they would be in most glass cases, and the cost usually is but a fraction of that necessary to procure glass museum cases of the usual design.

Since few institutions feel justified in providing a full-time curator for the museum, the task of maintaining the collection and the responsibility for its expansion usually falls on some one in the department of pathology whose time already is fully occupied. This often causes the collection to be neglected, which would not happen if the importance of the museum to the entire veterinary group were better appreciated. Unless strictly supervised, the assistance of students usually is unsatisfactory in the preparation of specimens, and while this class of labor can be secured at a relatively low wage, it often proves expensive in the end. Unless a regular curator is employed, the actual preparation of new specimens is best done by a member of the professional staff who is interested and the summer vacation period often can be used to advantage for this purpose.

While many more reasons might be advanced as to why the growth of the museums of comparative pathology has been retarded, the entire state of how good, bad or indifferent they are is dependent on the degree of enthusiasm evinced by those teaching. The museums may be dusty, ill-kept storerooms containing morbid curios, which eventually are discarded as worthless, or they may contain collections of properly preserved specimens so arranged and indexed as to make them usable exhibits commensurate with the ideals which represent the best in museum management. When the real value of a practical teaching museum is appreciated, other obstacles, such as those mentioned, become relatively insignificant.

In closing, it might be well to point out the desirability of increasing the scope of usefulness of the pathologic museum by making it readily accessible to the intelligent lay public. It seems that much could be done in soliciting the sympathetic understanding of the layman in the general problem of control of disease through the medium of a well-arranged exhibit of tissues, which show the pathologic changes characteristic of such eco-

nomically important diseases as hog cholera and tuberculosis. If the collection is properly arranged and attractively labeled, it is sure to appeal to the non-medical visitor for reasons other than morbid curiosity. The general public is interested in disease, and once it has been given the opportunity of obtaining a visual impression of the various ailments which affect live stock, the response, as measured in subsequent coöperation and helpful understanding, should more than justify the "open door" policy of permitting the public to visit the collection. I do not mean to infer that the public now is generally denied this privilege by most institutions. I make the point, however, that although such visitation is possible, the opportunity is taken only by few, owing to a hesitancy on the part of the public to invade what may be forbidden ground. By dignified means, proper notice could be given that visitors would be welcome and the museum, instead of being a darkened, unused institution, except on special occasions when students use it, would become an important instrument in the furtherance of desirable public relations.

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DISCUSSION

DR. H. C. H. KERNKAMP: Having had the privilege, more than once, of visiting the medical museum at the Mayo Foundation, it is easy for me to see how Dr. Feldman becomes so enthusiastic over medical museums. I know that anyone in this group who would take the time to go there and carefully inspect their museum would find it most helpful and interesting. The museum is splendidly arranged and the specimens adequately described, so that one can well appreciate the entire pathological process that has gone on in the particular specimen.

DR. E. T. HALLMAN: How is your system of preservation coming out? You are using the vacuum system, are you not? How are the specimens?

DR. KERNKAMP: They are holding up very well. I have some, four and a half years old, that are as good today as they were when I put them away.

DR. J. TRAUM: Does anybody know of a system whereby alcohol could be eliminated?

DR. KERNKAMP: Yes; the one we use.

DR. TRAUM: It is reported somewhere?

DR. KERNKAMP: In the *Bulletin of the International Association of Medical Museums* (Bulletin No. 11, 1925, and Bulletin No. 12, 1929).

DR. TRAUM: Why don't you add a little note to the reference of this paper as an answer to my question? Many of us are handicapped by the alcohol dished out to us in pint lots.

DR. KERNKAMP: No alcohol is used in this preparation.

DR. TRAUM: Do you remember what the method is?

DR. KERNKAMP: No; I do not. However, I am of the opinion that in addition to the Kaiserling formula, chloral hydrate is added. I have been more or less acting in the capacity of curator, yet do very little of the technical work. We have been fortunate in obtaining the services of a man who is working for us on a part-time basis, and is particularly adapted to this work. It is my job to collect the specimens and select the part or parts best suited for display. The methods of mounting are described in the bulletins referred to earlier.

DR. HALLMAN: Some one might be interested in the system we have been using for about fifteen years. I don't understand why I have not seen it in use in this country. This was published in about 1913 or 1914, in the *Journal of Comparative Pathology and Bacteriology*. I have not tried your method because I like the one we have used for fifteen or sixteen years so well. The preparations are fixed in formalin, according to the Kaiserling method, and run through the alcohols. Then for a few days they are placed in an arsenious acid-glycerin solution. I cannot give you the proportions. After remaining in that arsenious acid-glycerin solution for a few days, they are embedded in a solution with 10 per cent of gelatin added, and I have never tried any method that preserves color as well as this method. We are entirely satisfied. The only objection is that occasionally the gelatin will liquefy. That is no objection to the method.

I think it is a little better than your method. I don't say that is shows up better, but it entails less effort than the vacuum method, and I have no desire to change. I don't know why it is not used more generally.

DR. TRAUM: You cannot handle them for teaching so well.

DR. HALLMAN: Yes, we load them on a tray and take them into the classroom.

DR. TRAUM: Do you take them out of the jars?

DR. HALLMAN: No; you cannot take them out, but I do not think that is advisable anyway. This glass is full of gelatin. Students, in handling the specimens, if they happen to knock the top off, do not spill your solution. If a student knocks the top off one of these jars, no harm is done because the medium is solid and does not spill.

DR. KERNKAMP: I agree with Dr. Feldman that once a museum is begun, it will interest you and cause you to go further with it. You may want to display specimens depicting developmental stages of a disease, etc. With the method we are using, the specimens are collected and placed in a first solution consisting of potassium acetate, 85 gms.; potassium nitrate, 45 gms.; chloral hydrate, 80 gms.; formalin, 444 cc, and water 4000 cc. They are left in this until thoroughly fixed, from 6 to 48 hours, depending on the size of the specimen. The specimen is then removed, surfaced, and placed in running water until all the formalin has been removed, when it is put into a second solution consisting of potassium acetate, 26.6 gms.; chloral hydrate, 13.3 gms.; glycerin, 400 cc, and water, 3600 cc. In this solution the specimens remain as long as desired. We are now building glass frames to which the specimen is sewed before placing in the jar for final mounting.

DR. HALLMAN: I think we have some mimeographed sheets of the formula for this process, and if any one is interested and would write to me at East Lansing, I would be glad to send him a copy. It would save your going through the files.

RECENT PROGRESS IN DUCK DISEASE STUDIES*

By PAUL A. SHAW, *San Francisco, Calif.*

Toxicologist, California Fish and Game Commission

INTRODUCTION

For many years a malady, commonly known as the duck sickness or duck disease, has affected migratory water-fowl in certain marsh lands and shallow water areas located in the western states. Mortality has reached alarming proportions in many instances, and to determine the cause, and institute preventive measures, is an important conservation problem.

SPECIES, AREA, NUMBERS AFFECTED

Many species of shore birds and geese, together with ducks, are subject to this malady. Wetmore, in his report from Utah, listed 36 species of birds that he found suffering from the disease. The heaviest losses, however, are confined to ducks of the pintail, mallard, spoonbill, and teal species.

Focal points of the disease are located in the Bear River marshes and vicinity, of Utah; the Tule Lake and Buena Vista Lake districts of California; Lake Malheur in Oregon; and other areas of lesser importance in Idaho, Texas, Dakota and Canada. More recently the sickness has appeared at a number of gun clubs near Pond, California.

Dr. E. W. Nelson, former chief of the U. S. Bureau of Biological Survey, estimates that 10,000,000 ducks were lost in Utah up to 1927, and that 15,000,000 were lost in the western states as a whole. These figures are for ducks alone and should be doubled to include other species of birds. During a severe epizootic on the Bear River in 1912, there were gathered and buried between August 22 and September 21, the bodies of 44,462 ducks. However, in many instances, there are outbreaks involving only a few birds. In any case it appears to be a relatively small percentage of the birds in an area that are affected.

TELLURIC CONDITIONS

The sickness ordinarily occurs during the hot and dry period of late summer and fall, and is associated with the low water, decomposing vegetation, and high salt concentration of that

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season. The majority of disease areas are poorly drained, and in the past few years there have been marked decreases in the water area available for feeding and loafing. These decreases are the combined result of dry years and irrigation projects which have diverted water to other interests. In many instances the disease has been noted on recently flooded land, such as a field flooded by a broken dike, or on commercial gun clubs where new land is flooded to attract the birds. In most localities the sickness appears annually, but is more virulent at intervals of several years. An epizootic is usually terminated by heavy rains or in some instances merely by a drop in temperature without rain.

Symptoms

In a large part the symptoms of duck sickness indicate a paralysis of the nerve centers controlling the muscular system. This is shown by progressive paralysis that first renders the bird unable to fly and later spreads to the legs and neck. Whether the symptoms are due to a marked weakness or a true paralysis is hard to say. In many cases the nictitating membrane or third eyelid will not function or its response is greatly reduced. The bird gradually sinks into a completely helpless condition, which may persist for several days. However, the bird is usually alert, and many birds unable to fly can still paddle rapidly through the water and escape. Other birds die from drowning when the neck becomes too weak to support the head.

Yellow to greenish diarrhea is ordinarily present, and in the later stages this may harden to a cement-like mass, encrust the feathers around the anus and even clog the rectum. There is often a watery discharge from the eyes and nostrils, which may gradually harden, cement the eyelids together and even fill the throat, causing suffocation. Respiration is slow and difficult and the body temperature is subnormal.

Postmortem examinations indicate the birds to be fat or at least in good condition. An emaciated specimen is rare. The gizzard is empty except for gravel and seeds not readily digestible. The intestines are somewhat irritated, shrunken, and contain a thick yellowish mucus; the cloaca in many specimens is distended. There is a marked sloughing of the gizzard-lining at the point where it joins the proventriculus. The gall-bladder is enlarged and the intestines and gizzard are often bile-stained. In general the other organs appear normal.

The bird may remain in a helpless condition for 2 to 4 days, after which improvement is rapid. Figures indicate that 70 to 90 per cent of afflicted birds will recover when given fresh water and good food.

Theories and Statements Regarding Cause of Disease

Studies conducted on this disease have served to indicate that a toxic chemical, either organic or inorganic, rather than an infection of a bacterial or parasitic nature, is responsible. Wetmore, in his report on the duck sickness in Utah, states that the disease is caused by ingestion of water containing the soluble salts, magnesium and calcium chlorid. This explanation, however, has not been fully accepted by others studying the problem. Theories have been advanced that trade wastes from smelters and sugar refineries are to blame. However, many disease centers are located far from any industrial activity, and it is evident that a disease of such widespread character and intensity could scarcely be the result of such agencies. Others have intimated that gases formed during the decay of organic matter may be responsible. If this be true, it is not yet backed up by sufficient experimental evidence. It is still the popular theory that alkaline waters are the causative agent.

Organization of Work in California

Numerous studies have been undertaken by the California Fish and Game Commission in an effort to solve the mystery. At present the work is arranged under a cooperative program between the University of California and the Fish and Game Commission. Laboratory facilities have been provided in the Hooper Foundation for Medical Research, with Dr. K. F. Meyer, Director of the Foundation, supervising the studies. The investigation of duck sickness is one of many problems being given consideration.

Field, Laboratory and Experimental Findings

Parasitism: During the latter part of 1927, Mr. E. C. O'Roke made a survey of parasitism in ducks and geese from the San Joaquin Valley. The study for that period disclosed no apparent relationships existing between duck sickness and the presence of parasites. Parasites were found in a large percentage of the birds examined, and were found in nearly all the species commonly taken during the hunting season. With but two or three exceptions, the parasitized birds seemed to be in as good condition as the unparasitized.

Poisonous seeds: During an outbreak of the sickness in 1928, Mr. McLean, naturalist for the Division of Fish and Game, examined the stomachs of birds taken by the hunters and also of diseased birds. The study revealed no seeds of a poisonous nature and only one plant of a poisonous species was located in the area. This was the black nightshade. Mr. McLean concluded that the probability of seed poisoning was remote, as some of the birds found sick were not seed-eaters.

Bacterial infection: In the fall of 1928, Dr. H. Van Roekel carried on investigations for a period of six weeks at the Hollywood Gun Club, near Pond, California.

Attempts to culture a causative organism from the tissues and intestinal tract of diseased birds gave negative results. Extracts obtained from the organs of diseased birds were injected into healthy birds, and in no case was the malady transmitted by this method.

Gross pathology was studied on a large number of the diseased specimens, with general findings in agreement with those previously described. Recovery data also were in agreement with those found in previous investigations.

One experiment was conducted by holding recovered birds in a pen located on a pond suspected of producing the disease. While a few of the birds on this experiment died, it was felt that their death was due to unfavorable environment, rather than to reproduction of the disease.

Chemical studies: Chemical studies are in progress along several lines, some interesting results having been obtained in the study of blood chemistry. Four blood components were determined on 15 diseased and 13 normal ducks of the pintail species. Non-protein nitrogen was found to average 50 per cent higher in the diseased birds and uric acid averaged 80 per cent higher than in the normal birds. No striking differences were to be noted in chlorids or blood-sugar.

Several normal birds were given suspected water to drink for a period of ten days. For one series the water was concentrated to one-fourth the original volume for the experiment. All these birds remained healthy and the blood tests at the end of the period did not show the changes characteristic of the diseased birds.

An extended field and laboratory program, based on the possibility of some type of salt poisoning, is under way. This study may be divided under three headings:

1. Experimental feedings to determine the effect of single and mixed salts.
2. Soil and water analyses from both disease and non-disease areas.
3. Experimental feedings, using natural waters, or water extracts of the soils collected.

Data on the toxic and lethal doses of certain salts have been obtained. Sodium chlorid, a relatively inert salt, has been found lethal for ducks in amounts of six grams or more. Six grams caused death in four days; 8 grams caused death in 40 hours; and 16 grams in 2.5 hours. A torticollis and depression, rather than the paralysis of duck sickness, was observed.

Magnesium chlorid in amounts up to 1 gram produced no noticeable toxic symptom, whereas 1.5 grams produced a paralysis that began in 40 minutes and lasted 5 to 6 hours. The bird remained alert, but had no control of the legs, wings or neck. Recovery was apparently complete by the following morning. Additional amounts up to 3.6 grams produced similar symptoms, a little more acute, but not lasting for a longer period. A dose of 4.5 grams caused paralysis in five minutes and death in 15 minutes. A 2 per cent solution, given as the drinking water for a one-week period, was apparently non-toxic.

Calcium chlorid amounting to 4.8 grams caused depression in 15 minutes and death in 35 minutes. Reduction of the dose to 1.4 grams removed all toxic symptoms. A 2 per cent solution, given as the drinking water, produced no symptoms resembling the duck sickness. One bird on this experiment died in two days; the other remained healthy during a 7-day experiment.

Sodium sulfate totaling 5 per cent in a mash food was fed for one week. This produced a marked weakness and diarrhea, followed by death in two weeks. The symptoms resembled the duck sickness in some respects. A single dose of 2.6 grams of this salt was without effect.

A 1 per cent solution of sodium bicarbonate, given as the drinking water, caused wing and leg paralysis, followed by death in 5 to 7 days.

Some specially interesting results have been obtained through the feeding of salt mixtures. It has been found that 1.5 to 2 grams may be lethal if the mixture contains 5 to 8 per cent of nitrate, together with smaller percentages of either bicarbonate or magnesium. These mixtures contained sodium chlorid and sulfate as the bulk of the dose. Toxicity was further increased

by small amounts of nitrite, and by a substitution of potassium for a portion of the sodium. Leg and wing weakness, together with a discharge from the eyes and nostrils, were the chief symptoms noted. These feedings caused death in 5 to 10 hours, or were followed by recovery in less than a day. However, in those feedings containing nitrite the symptoms were maintained for a longer period.

It is possible to delay toxicity from 2 to 5 days by giving similar mixtures of lower concentration for the drinking water. Symptoms more typical of the disease are developed by this method than by force-feeding larger amounts for a shorter period. However, the percentage recovery following such treatment is extremely low and, at the same time, the helpless condition does not appear quite so acute as sick birds that are observed in the field. Postmortem examinations on these experiment birds bear marked similarities to findings in diseased specimens.

A large number of soils have been collected and analyzed to determine major differences in the inorganic ions present in soils of disease and non-disease areas. A study of the data obtained is in accord with the data on salt mixtures; the soils from disease areas showing a high relative percentage of nitrate and nitrite; and the soils of non-disease areas showing a small percentage of these ions. The study also indicates that sodium chlorid and sulfate compose the major increase in soils of high salt concentration. No major differences were to be noted in the other ions studied.

Experimental feedings using soil extracts, varying from 1 to 10 per cent total salts, have produced symptoms that were typical of those obtained through feeding mixtures of the pure salts.

Recently there was found a nitrate soil of exceptionally high toxicity. One hundred cc of a water extract containing 1 per cent total mixed salts was fed over a two-day period and caused death in both birds on the experiment. Thus but 1 gram of mixed salts was lethal in this instance, and toxic symptoms were to be noted when but half of the amount had been given. This experiment was conducted under the San Joaquin Valley summer temperature of 100 to 110 degrees, and this factor may possibly have influenced the toxicity. A check on this experiment will be made in San Francisco to determine whether temperature or a more toxic mixture was responsible for the results.

It is of interest that areas of shallow water, high in soluble salts, have been located during all outbreaks of the disease so far

studied. In this regard it might be questioned whether a duck would accept water and food of high salt content. It has been found, however, that a duck will drink highly saline water quite readily, and mash food containing up to 15 per cent total salts has proved acceptable.

Preliminary experiments to determine the toxicity of hydrogen sulphid, indicate that 0.02 per cent in the air is a lethal concentration for ducks. This treatment causes a muscular weakness and affected eyes, but also causes a pronounced gasping, which is seldom noted in sick specimens.

We feel that the findings regarding nitrate soils and the toxicity of nitrate mixtures are specially significant. The nitrate is approximately four times more soluble than the chlorid and sulfate. For this reason one would expect this salt to become an appreciable factor in the surface soil only at the hottest and driest time of the year. In addition both the nitrate and nitrite are products of organic decomposition. These facts correspond with the usual incidence of the disease in late summer and in areas of organic decomposition and high salt concentration. However, we are not satisfied that the identical picture of the disease has been reproduced and there are many considerations yet to be cleared up.

Discussion

Dr. J. C. Hargrave: Have you had any experience in regard to wild ducks?

Mr. Shaw: Not that I know of. That particular end of the work has been handled in the past by Dr. Van Roekel, who is now in Massachusetts, and I really would not be able to answer that question, although I don't think so.

Dr. Hargrave: In the province of Alberta, particularly in the southern region, we have had in the last few years tremendous losses in wild ducks, and it is said to be due entirely to a blood-sucking parasite and they have very much the same symptoms as you have recited. When these parasites were removed, nearly all the ducks recovered. Nothing else was done except to remove the parasite.

Dr. J. N. Shaw: Dr. W. T. Johnson has found a leech in the ducks from Tule Lake, that were sent up to Corvallis for examination during the time the ducks were dying. The leeches were in the proventriculus.

Dr. W. J. Butler: Have you had much of that trouble in Canada?

Dr. Hargrave: As far as I know, it has been confined to Alberta and probably to southwestern portions of Saskatchewan, where conditions are similar, and where they have a large percentage of coulees, although the ducks have gone over to fresh water lakes and the losses have been extremely large.

Dr. Butler: Mr. Shaw, in your experimental studies on these animals, have you starved them before you gave them the magnesium sulfate?

Mr. Shaw: No, not necessarily.

Dr. Butler: Under natural conditions, have you found that the animals have just undergone rather a long flight before they became sick?

Mr. Shaw: That has been true in a good many instances. We have had that reported in a number of instances, soon after they have come from a distant area.

Dr. Hargrave: I believe a good many leeches are present.

MR. SHAW: I wouldn't say that entirely. I think work of that sort can still be carried on. In that paper I intended to give more of the results of our chemical studies. We will soon have a man with us who will be studying that phase of the work to a greater extent, Dr. and Mrs. Hobmaier, who will work in the Division of Fish and Game.

DR. SHAW: Did the birds recover after experimental treatment with the salt solution?

MR. SHAW: It depends on the regularity. If they are given a large amount of salt mixtures over a short period, recovery is likely to take place in a short time. If it doesn't kill them, they recover rapidly. However, lower concentrations over a longer period, say five or six days, or a week, do not permit so good a recovery.

DR. SHAW: Have other birds been found to be affected besides the geese?

MR. SHAW: Yes, several other species.

Supreme Court of Iowa Upholds Tuberculosis Eradication

In a 16-page decision, rendered September 22, 1930, the supreme court of Iowa sustained the law under which the eradication of bovine tuberculosis is being conducted in the State. The case before the court resulted from the efforts of certain cattle-owners in Mitchell County to obtain an injunction against the state and county authorities to prevent the tuberculin-testing of cattle. In rendering its decision, which in every particular was favorable to the continuation of tuberculin-testing in Iowa, the supreme court reversed the opinion of a local judge in Mitchell County. "Careful reading," the decision stated, "convinces us that the test is reliable, useful, and advantageous." The supreme court also upheld the view that bovine tuberculosis can be transmitted to human beings.

The decision is considered by veterinarians to place a highly constructive legal interpretation on tuberculosis-eradication work. This decision is in accordance with other similar decisions concerning the reliability of the tuberculin test and its application in the uniform plan by which the states and the federal government are steadily eradicating bovine tuberculosis.

Third of U. S. Practically Free from Bovine Tuberculosis

More than one-third of all the counties in the United States are now practically free from bovine tuberculosis, the U. S. Department of Agriculture announced the past month. On October 1, there were 1,035 counties classified as "modified accredited areas." A high mark in this work was reached in September, when 24 counties were added to the modified accredited area. These counties were located in nine states and included a total of 432,163 cattle. Twelve of the counties were in Ohio, where very rapid progress recently has been made in the campaign.

RECENT ADVANCES IN THE PROPHYLAXIS AND TREATMENT OF CANINE DISTEMPER*

By F. W. Wood, *Berkeley, Calif.*

Since the publication of the report[1] on the cause and prevention of dog distemper by Laidlaw and Dunkin, in 1928, but little new information on canine distemper has found its way into the literature. The above-mentioned authors showed conclusively that canine distemper is a specific filtrable-virus disease, and their work has been amply confirmed. In the report of the A. V. M. A. Committee on Distemper for 1929 the chairman states: "I do not think there is any doubt that the cause of canine distemper is a virus." This opinion is held by practically everyone at this time.

Among the animals susceptible to canine distemper, which have been reported, are the dog, fox, ferret, weasel and stoat. In addition to these it may be reported that mink seem to be as susceptible to distemper virus as are ferrets. On April 1, 1930, two sick mink were brought to this laboratory at the suggestion of Dr. Turver, a practicing veterinarian in Oakland. The history obtained from the owner was that his dog had died some two weeks previously with what he thought was distemper and that this dog had been kept in close proximity to the mink. On April 2, the mink was found dead and an autopsy was performed and the spleen removed. On April 4, a ferret was injected with emulsified spleen from the mink. The ferret stopped eating on the 12th, was noted visibly sick on the 14th and died on the 18th of April, with what appeared to be a typical case of distemper.

On April 2, a portion of the spleen obtained from the mink was given to Dr. E. T. Dewey, Department of Bacteriology, Stanford University, who injected a young dog with a portion of the emulsified spleen. Dr. Dewey stated that this dog developed distemper after the usual incubation period and later died. On May 15, through the kindness of Dr. Dewey, three normal mink were received at the laboratory. On May 16, two of the normal mink were inoculated with canine distemper virus 172, the third mink being held as a control. Both of the mink receiving injections of the virus were found dead on June 2, sixteen days after inoculation. The control mink was apparently normal at this

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date and was then inoculated with canine distemper virus 175. This animal died of what appeared to be typical distemper infection, June 17, fifteen days after inoculation. This report of the susceptibility of mink to distemper virus is included in this paper as a matter of interest, since mink have not previously, to our knowledge, been considered among the list of susceptible animals.

In their report for 1928, Laidlaw and Dunkin outlined a method for the production and administration of two products intended for use in the active immunization of dogs and ferrets against distemper. The first of these products was called a vaccine and was prepared from the spleen of dogs infected with the filtrable virus of distemper. The method of preparation is as follows:

> Ferrets suffering from acute distemper are killed by an over-dose of chlororform on the fourth or fifth day of illness, when they are usually *in extremis*. The carcass is dipped in 2 per cent lysol and pinned out. The spleens are removed with sterile instruments, using strict aseptic precautions. The spleens are weighed, pounded in a sterile mortar and rubbed up into a smooth mass as quickly as possible. Sufficient saline is added to yield a 20 per cent suspension of spleen pulp and after brief stirring the contents of the mortar are transferred to a sterile bottle containing glass beads. The bottle is shaken vigorously in a machine until all the fragments seem to be broken up. The contents of the bottle are then filtered through a double thickness of sterile butter-muslin into a sterile bottle. The shreds of tissue are thus removed and discarded. A small quantity of the filtrate is removed for potency test. Sufficient strong formaldehyde solution is added to the main bulk to yield a final concentration of .1 per cent, after which the mixture is shaken and transferred to the cold store. After four days the formolized spleen suspension is invariably non-infective for ferrets, and sterility tests are made both for aerobic and anaerobic bacteria. If the product proves to be sterile, as is usually the case, it may be used as a vaccine without any further treatment. Owing to the presence of the small amount of formaldehyde, however, vaccines of this kind are irritant when injected subcutaneously. A non-irritant product can be made by adding a sufficient ammonia solution to give a faintly alkalinic reaction. The ammonia is added until a reaction of pH 8.0 to pH 8.2 is secured and thus all free formaldehyde is converted into urotropin and the final product is devoid of irritating properties.
>
> For the potency test referred to above, a 20 per cent suspension of spleen pulp is diluted freely and doses corresponding to 1/10,000 to 1/100,000 gram of spleens is injected into two ferrets. Both animals should sicken with distemper within ten days. If they do not do so it is clear that the virus content of the material which was converted into vaccine was low, and the resultant vaccine will probably prove to be inferior. Owing to the long incubation period of distemper in the ferret, it is necessary to assume that the material will yield a good vaccine and finish the preparation of the vaccine at one sitting. It is not advisable to keep the ferret spleen while the potency of these organisms is being determined, as during the wait of ten or more days considerable deterioration will occur in the virus content of the tissue

The second of the products used for immunization purposes, according to the Laidlaw-Dunkin method, consists of distemper virus. This is obtained from spleens of ferrets during the height

of the infection, the spleens being finely ground and quickly dried *in vacuo* at a low temperature. Two weeks after the administration of the vaccine or antigen, treated animals should receive an intradermal, or, as is often used, a subcutaneous, injection of a small amount of the virus. After a lapse of about ten days or two weeks following the administration of the virus, treated animals are solidly immune to distemper.

The preparation of the dog distemper vaccine is the same as that outlined for the ferret vaccine with the exception of the fact that dog tissue is employed instead of ferret tissue, it having been found that homologous tissue is more effective as an immunizing agent than heterogeneous tissue.

Virus Modified by Several Agents

Laidlaw and Dunkin have stated also that storage, heating and carbolization modify distemper virus tissue, resulting in the production of active immunizing vaccines or antigens. They also have stated that in their hands vaccine or antigen production was more uniform and satisfactory where formalin was employed as the modifying agent than the methods just mentioned.

Laidlaw and Dunkin cite extensive and successful experimental use of their method of immunization on practically all breeds of dogs and suggest that their method of immunization can be used generally for the prevention of distemper infection among susceptible dogs, and following their suggestion their method of immunization has been widely employed during the past two years both in England and the United States.

The experimental results of the immunization of dogs in England were apparently very successful and the percentage of reported successful immunizations of dogs against distemper was given as about 99 per cent.

Comerford[2] gives an enthusiastic report on the use of the Laidlaw-Dunkin method from the standpoint of a practitioner, but pointedly cautions veterinarians to be very careful in the selection of dogs for the treatment. He states:

> If there is any suspicion that the puppy is not fit, postpone treatment for a day or two. Handled properly we have the whole canine population in the hollow of our hand, but it all depends on individual effort. One failure at the beginning is a serious set-back. If you are unlucky enough to experience bad luck following an inoculation, don't take it for granted that the material is at fault. Go into it with a perfectly open mind and you will find the true cause of the trouble and vindicate the honor of the treatment.

Laidlaw and Dunkin also emphasize the need for the careful application of their method of immunization, laying particular stress on the fact that only normal dogs should be immunized and that immunized dogs should be carefully protected against infection during the immunization period. As was to be expected following the distribution of canine immunizing agents on a large scale, results from the treatment did not approximate the experimental reports of the originators of the method and many veterinarians, as well as dog-owners, have been greatly disappointed following the use of the immunizing treatment.

A recent issue of the *Veterinary Record*[3] contained the following editorial, which well illustrates the natural disappointment which followed the failure of the Laidlaw-Dunkin method to live up to expectations created by the reports of experimental work.

> We learn that Messrs. Burroughs, Wellcome and Company have decided to withhold supplies of both dog distemper vaccine and virus for the time being. This announcement will doubtless be received with some astonishment and much regret by many members of the profession, but, as the distribution of these products has been confined to veterinary surgeons, this early announcement informing the profession of the decision of Messrs. Burroughs, Wellcome and Company is deemed desirable.
>
> Although the first issues were very effective, we understand that more extended experience of large-scale production and distribution of the prophylactic has revealed certain difficulties which have caused serious disappointment to some members of the profession, and until these difficulties can be overcome it has been considered advisable to stop the issue of the product.
>
> It will be readily understood that, with a highly unstable product such as dog distemper virus, it is exceedingly difficult to be certain that supplies of guaranteed quality will be available at any time. It is now certain that the virus is more unstable than it was originally thought to be and there is reason to believe that on some occasions the virus has lost potency in transit to the veterinary practitioner, with the result that the full inoculation course has been incomplete and some hounds and dogs have been imperfectly immunized. These instances, believed to be a small proportion of the whole, have caused much trouble and dissatisfaction to practitioners and dog-owners and the scientific staff engaged at the Wellcome Physiological Research Laboratories are busily investigating the conditions necessary to ensure that this product shall reach the veterinary surgeon in the same state of potency as when it was dispatched. Additional and more stringent tests to control the protective properties of the vaccine are also, we believe, in hand.
>
> It is not known how long veterinary surgeons will be required to wait before supplies become available again, but it is confidently hoped that two or three months hence will see the resumption of distribution.
>
> Much depends on the results of the work contemplated by the scientific staff of the Wellcome Physiological Research Laboratories, but we believe that the difficulties will soon be overcome, and in expressing the hope that in the near future we shall be able to announce a resumption of distribution of reliable and potent agents which the veterinary surgeon may, with confidence, recommend to his clients, we are expressing the feeling of every member of the profession.

This editorial expression nicely states the situation as it stands today as regards the successful immunization of dogs against distemper. While admitting results have not met expectations, faith is expressed in the ability of the investigators and producers to meet the difficulties so far encountered and successfully overcome them.

Canine distemper virus is a very delicate product, which can be maintained in a viable state, outside the body of the infected animals, only with great difficulty and this fact is one of the serious obstacles which has so far prevented the successful application of the vaccine or antigen virus method advocated by Laidlaw and Dunkin. Virus can be maintained under proper storage conditions, with regularity, but when this product is exposed to field conditions, such as shipment and handling, rapid deterioration may follow, and the administration of inert virus is, of course, a useless procedure as far as establishing an effective immunity goes and leads only to disappointment on the part of the users. For the reasons just stated some of the producing laboratories in this country have so far refrained from attempting to supply canine distemper virus. Again, in the case of the vaccine or antigen we have a product which may be prepared from several tissues and one in which deterioration may occur following preparation, due to improper handling during distribution. The vaccine or antigen is more stable than the virus, but nevertheless is subject to loss of immunizing properties if kept under unsatisfactory conditions.

Different Tissues Employed

In the preparation of canine distemper vaccine or antigen several tissues may be employed. Among these tissues may be mentioned spleen, lymphatic glands, liver, brain and spinal cord. The immunizing properties of the vaccine are apparently dependent on their original virus content, and the virus content has been found to be most constant in the splenic and lymphatic tissues, and practically all investigators are in accord with the statement that the most uniform and satisfactory vaccine is prepared from splenic and lymphatic tissues. With reference to the selection of tissues for the preparation of the vaccine or antigen, the original workers, Laidlaw and Dunkin, state that they have found the brain and liver to produce efficient vaccines. In their hands, however, they abandoned the use of brain tissue, since the source of supply was relatively small, as well as the

liver, by reason of the fact that vaccines prepared from liver were variable in their immunizing properties and that it was difficult to prepare a sterile product from a vaccine containing liver tissue.

Boynton,[4] in his original work describing the production of a rinderpest vaccine, states that although the liver, heart, kidneys and blood contain abundance of virus, they are not suitable tissues when used separately or collectively; but, when added to the lymphatic tissues and spleen, they form a vehicle which increases the yield of the vaccine to a marked degree.

Kelser,[5] working with a modification of Boynton's rinderpest vaccine, points out that if a single-injection vaccine is to be employed for the immunization of carabaos, "it appears desirable, in the preparation of a vaccine for such animals, to use only tissues of very high potency (lymph-glands and spleen)." Rodier[6] continued Kelser's work and demonstrated that a rinderpest vaccine prepared from lymph-glands, spleen and tonsils was of such high immunizing value that a single dose of this vaccine would protect cattle and carabaos against virus inoculation.

It is the writer's belief that spleen and lymphatic tissues from properly selected virus dogs will produce a more uniformly safe and potent vaccine or antigen than can be prepared from other known sources.

Preparation of Homologous Serum

Anti-canine distemper serum (homologous) is prepared from the blood of healthy dogs immune to distemper which have been hyperimmunized by injections of a massive dose or doses of virulent distemper virus. This serum when properly prepared is a sterile product, has good keeping qualities and is useful for the passive prophylactic immunization of susceptible dogs against distemper infection as well as for the treatment of infected dogs during the early stages of the disease. Best results from the use of serum, both as a prophylactic and as a therapeutic agent, are to be expected when the serum is used in full dosage.

For the active immunization of dogs against distemper by the Laidlaw-Dunkin method, two products are used: the vaccine, or antigen, and distemper virus. The dose of the vaccine is 5 cc and injections are made subcutaneously. All precautions as regards asepsis should be observed when injecting the vaccine and it is good practice to divide the dosage, as well as to make injections well under the skin, so as to assist absorption. Only

healthy dogs which have not been exposed to distemper should be given the treatment, and all treated dogs must be safeguarded against exposure for at least one week following treatment. These precautions are necessary, since a period of about ten days is required for the development of immunity following the administration of the vaccine or antigen. Ten days to two weeks after injecting the antigen or vaccine a small dose of virus is injected intradermally or subcutaneously. Two weeks after giving the virus, treated animals should be immune to distemper. This method of immunization is very satisfactory when it is safely completed (that is, when a potent antigen and a virulent virus are used), but it is one which is open to several objections from the standpoint of the average practicing veterinarian.

Difficulties Encountered

Among the difficulties encountered by the veterinarian when using the Laidlaw-Dunkin method of immunization may be mentioned the practical impossibility at times of determining whether or not the dog presented for immunization has been or will shortly be exposed to distemper. Dogs in the incubative period of distemper show no symptoms of infection whatever. A second difficulty is that of preventing exposure during the ten-day period following the immunization treatment. This factor very often presents difficulties which are very hard for practitioners to control. A third factor to be considered is that of the degree of immunity which is conferred by the administration of the vaccine or antigen. If the immunity resulting from this treatment is not good, the administration of virus will at times provoke an attack of distemper. Results of this kind, that is, dogs coming down with distemper following administration of the virus, are a source of worry and are very hard to explain satisfactorily to owners. The fourth, and probably the greatest difficulty to overcome with the vaccine, or antigen, and virus immunization is in being certain that the virus is of sufficient virulence at the time it is administered. As has been stated previously, great difficulty has been encountered in preparing canine distemper virus in a stable form, and this product is apt to lose its virulence suddenly, and there is no practical means at present of determining whether a virus is active at the time that it is administered.

Where the vaccine or antigen is used alone for active immunization purposes, dogs are injected in the same way and with the

same product as that described for the first injection of the regular Laidlaw-Dunkin method. Treated animals are injected with 5 cc of canine distemper vaccine or antigen, injections being made well under the skin and observing all precautions as to asepsis. Following the administration of the vaccine or antigen, dogs should be protected against exposure to distemper for at least ten days and care must be taken to ascertain if the treated dogs are healthy at the time the treatment is given. The same objections as were outlined under the preceding method of immunization exist also where the antigen or vaccine is used alone, except, that since no virus is employed there is no danger of breaks as exist where virus is given following the vaccine or antigen treatment. The immunity following administration of vaccine or antigen alone is not so high nor so lasting as would be expected where virus is used. The duration of this immunity is usually placed at about four months, unless treated dogs should be exposed to virus infection during the period of immunity. In cases where exposure does occur, a lasting immunity is conferred similar to that which may be expected following the administration of a potent virus.

Serum-Virus Treatment Promises Best Results

The combination of homologous anti-canine distemper serum with a vaccine or antigen, followed after an interval of two or more weeks by a second and even a third injection of antigen, seems at the present time to offer the most practical means for the immunization of dogs against distemper. When giving the immunizing treatment, using serum in combination with vaccine or antigen, 10 cc, or preferably more, of homologous anti-canine distemper serum is given with the first dose of antigen. The injection of both products should be made subcutaneously, using the usual precautions as regards asepsis. The serum is given for the purpose of modifying the effect of exposure to distemper previous to treatment, as well as to safeguard animals for the ten-day period following treatment, so that if dogs are infected at the time, or soon after, the treatment is given, they stand a very much better chance of making a good recovery. The second and third injections of vaccine or antigen are advised for the purpose of increasing the immunity response and producing a longer period of immunity than would be expected following a single injection of the vaccine or antigen. It is believed that this method of immunization *is* the safest and most workable plan to follow at the

present time and until both the vaccine or antigen and the virus can be more accurately standardized and prepared in more stable forms.

Should the time arrive when canine distemper virus can be prepared in a stable and dependable form, the use of virus with homologous anti-canine distemper serum should offer a most satisfactory means for the active immunization of dogs against distemper. The simultaneous injection of homologous anti-canine distemper serum in proper dosage, together with canine distemper virus, will actively immunize dogs against canine distemper infection. The only objection to this method at present lies in the fact that the distribution of the virus has not as yet proven satisfactory.

The diagnosis of canine distemper is not always easy and it is believed that in practice mistakes are often made as to what constitutes true filtrable-virus distemper cases. Infectious pneumonia of dogs, usually due to *B. bronchisepticus* infection, presents symptoms which closely simulate those associated with true distemper. There is also an infectious enteritis of dogs, a condition also difficult to differentiate from distemper. Quite recently a disease of foxes described by R. G. Green and E. T. Dewey[7] has been identified and it is possible that this disease may account for some of the cases in dogs classed under the heading of nervous distemper. There is great need for more investigational work on canine distemper and the writer believes that more information on canine infections will help to clarify the canine distemper situation.

Summary

Summarizing the results in the field, it would seem that for the present, at least, canine distemper virus has proven too unstable a product for general distribution.

The vaccine or antigen is a satisfactory and safe product for general use but the immunity is not of long duration.

If a stable form of the virus can be prepared for general distribution, the serum-virus method of immunization suggests interesting possibilities.

Improvement of the antigen or vaccine is a possibility and if this can be accomplished it will be the best product to use for the prophylactic immunization of dogs against distemper.

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DISCUSSION

DR. E. R. STEEL: In regard to the technic of injecting the spleen vaccine, we have worked out a little plan, which we use also for the injection of rabies vaccine.

There was a good deal of complaint, at first, about the formation of abscesses. So far we have had one abscess. That was in a little fox terrier which jumped around on the table, and we couldn't make the proper kind of an injection.

The method we use is simply to take the region of the scapula, where the skin is loose, and pull the skin forward as far as we can pull it, and then make the injection slowly, distributing the vaccine over an area of about six inches, by gradually releasing the skin. We do not make two injections. We have not found that necessary, either with the rabies vaccine or the distemper vaccine. When you become used to that method, if you will palpate that region after the injection, you will find no swelling there that could cause an abscess.

Just a word about the life of the spleen vaccine. I have gone over this work, in connection with the work of the A. V. M. A. Committee on Veterinary Biological Products, of which I have been a member for the last three years.

In reviewing the literature on the subject, I find that the British state that the vaccine may last from three to five months. I think the Bureau allows a dating of six months. We had some vaccine that had been kept in a refrigerator and would have been out-dated, according to that, in thirty days, but when we injected it we had a reaction following the second injection of the virus. The British state that the second injection of the virus is not dangerous. We had the worst break of distemper, I think, we have had in the hospital, in June, following the second injection in a dog, after we had used this long-dated vaccine. I think that a dating of ninety days probably would be preferable. The British state that the vaccine does deteriorate with age, although the keeping of the product is important, that is, if it is kept at the right temperature.

We injected three pups last year to see what we could do in the way of protecting them, giving them the spleen vaccine, and then in eighteen days the virus. One of the pups had a temperature rise of above 104° F. and was indisposed the fourth day following the second injection.

I will admit that a hospital is not the proper place to carry on experimental work with canine distemper, but we wanted to satisfy ourselves with some little pups given to us and we succeeded in keeping these dogs around for a couple of months.

We would assume that the injection should have been all right following the rise in temperature, at least in that one case.

Then we exposed the dogs, after two months, and two of the little, short-haired fox terriers, about ten days or two weeks after exposure in the distemper ward, acquired a cough, but didn't die. They were a little off, but not much. One of the students I had sent one home and I asked him how it got along. He said it was a little off for a few weeks, but pulled through. One of the dogs died within two weeks. I had sold it for $15.00. It went out of town, to St. Joseph, Missouri, and a veterinarian there said it had distemper two weeks afterwards. Evidently the virus in that case was not alive.

That is our experience with the vaccine. It did not protect much longer than two months when the dogs were severely exposed.

Regarding the life of the virus: I do not think that we can keep it much longer than four or five days. In fact, we are getting a virus that is claimed to be harvested the day previous. It is sent out packed in dry ice, special delivery. I know we have had some kick out of that virus.

We also follow the practice, in preparing a dog to be immunized against distemper, of first running the dog through the routine microscopical fecal examination and treatment for parasites and will not vaccinate a dog unless that has been done, to get rid of hookworms or other devitalizing factors. That dog is put on cod-liver oil for a period of two weeks and built up so that vitamin A is stored up in the liver, which we know is a disease-resisting vitamin.

We have done a little work in giving the vaccine in the hospital and have quit that, after one or two breaks, because it is a poor policy to give a dog vaccine and keep it in the hospital. During that period of ten days or so, following the vaccination, we have had a few breaks.

In regard to the serum: As far as I know, there has been nothing published regarding serum, much more than in a general way, by the British, to the effect that the serum may have some value if properly prepared.

I find, in investigating the proposition, that hardly any two houses make the serum the same. One house, in particular, was using the blood taken at the same time that the spleens and the lymph-glands were harvested and using that to hyperimmunize.

In the report of the British Committee, it is stated that the virus in the second stage of distemper is not uniformly found in the blood.

Then they are using an antigen of unknown value to produce a serum and they may not have a true distemper serum. They were not using the spleens or the adjacent glands, which were known to be uniformly full of the virus in the second rise. Another house was getting the blood in the early rise and injecting their hyperimmunes.

I would like to see, and I think the practitioners of the country are entitled to see, some definite experimental work on homologous serum published, so that it can be checked.

We have this disadvantage: In other animals our research laboratories and our state institutions receive appropriations to carry on research work because of the economic factor, while with pets we do not secure any money from our legislatures to carry on experimental work, so that we are absolutely dependent upon our commercial firms to carry on this research work for us.

I would like to see some more of that work actually published, so that we will know the exact method that is used and the results. (Applause)

DR. E. J. FRICK: I wonder if Dr. Wood would be kind enough to tell us the clinical symptoms of canine distemper in mink?

CHAIRMAN BOWER: Perhaps it would be better to make a note of all these questions and then you can have a rebuttal at the end.

DR. E. A. EHMER: First, in your opinion, Dr. Wood, what is the length of time this vaccine will be potent if it isn't kept on ice? I want to ask the same question about the homologous serum, and, if it would be possible to answer such a question, I would like to have Dr. Wood outline what he would consider the proper procedure, with the present material at hand, to immunize a puppy, we will say, from the time it is born. For instance, someone comes in and says, "I am going to have some puppies tomorrow. Tell me what to do from now on." You might consider that from the standpoint of either a toy or a medium-sized dog, or a larger dog.

DR. G. M. SIMMONS: Dr. Wood, would you be kind enough to outline the procedure for the hospitalization of surgical cases in order to prevent them from contracting distemper while they are in the hospital. Would you suggest just the injection of serum, or virus and serum, or vaccine alone? Do you consider it practical to educate the public to this point?

DR. D. K. COLLINS: Possibly the question that Dr. Ehmer asked will bring out the answer I am after. The question I have in mind is regarding the dosage of vaccine that should be given to dogs of various sizes, that is, whether or not it should be modified in some of the toys and possibly increased in some of the larger breeds.

DR. O. A. LONGLEY: Perhaps it would be better if I would talk after Dr. Wood answers these questions. I can offer some comment from a purely practical standpoint.

I have been intensely interested in immunizing young dogs for the purpose of hospitalization. Ever since the Laidlaw-Dunkin type of vaccine came out, I have used it extensively in my practice, not from a scientific standpoint, but from the standpoint of developing a practical, inexpensive method of immunizing all types of young dogs brought to the hospital for boarding, surgical cases, or for treatment of conditions other than distemper.

I believe we have tried every method that has been outlined, serum-virus, vaccine-virus, serum-alone, vaccine-alone, and every combination of those that could be conceived. After two years of practical experience, with from 40 to 75 immunizations a month, we have come to a definite system, and are following it successfully, and haven't had a break for a year. That is rather a broad statement, but it is a fact.

First, we endeavor to determine whether there has been exposure or whether the animal is showing any signs of distemper at all. As Dr. Steel has just stated, we make a clinical examination for evidence of parasitic infestations, although we do not go to the length of making bacteriological examinations, or microscopical examinations, although that is a good recommendation. I mean we have endeavored to simplify the practical method as far as possible and it has simmered down to this: Young dogs, regardless of age, after weaning time, receive a dose of homologous anti-canine distemper serum and a dose of the Laidlaw-Dunkin type of vaccine simultaneously. If they are to be in the hospital for only two or three days, they get the serum alone; if they are to be in for a week or ten days, or longer, they get the serum and vaccine. By vaccine I mean the Laidlaw-Dunkin type of vaccine. We are able not only to receive young dogs and to carry them for any period in the hospital successfully, but we can have litters of pups born in the hospital and immunize them shortly after weaning time without any difficulty whatsoever.

We had an experience in attempting to raise puppies under hospital conditions and lost a great number before the vaccine came out. Since the vaccine came out, and we have developed this method of immunization, we haven't lost a one or had a break. Others can do the same thing. It is as simple as can be.

A question was asked about dosage for the smaller breeds, Bostons, fox terriers, etc. We give 2 or 2.5 cc of the vaccine, and 5 cc of serum, that is, when it is administered soon after weaning time.

We know, from practical experience, that the pups born to mothers immune to distemper carry that immunity, apparently transmitted to them from the mother, for several weeks after weaning time. So we usually figure on immunizing the puppies born in the hospital about one month after weaning time. That would be pretty close to two months of age.

In order to keep our own young dogs immune during their entire period of susceptibility, which theoretically is close to two years, that is, to keep the dog immune until the time he is two years old, we give him a dose of serum and vaccine sometime between the ages of two and four months; two months after the first injection he gets a dose of vaccine alone; four months after the second injection, vaccine alone; and six months after the third injection, he gets his fourth and final injection which, I think, makes him about fourteen or fifteen months old. By following that method we have also been able to keep our clients' young dogs immune, as proven by the fact that we can take them in the hospital as boarders repeatedly during the first two years of their lives and have no break or a failure.

I have outlined this method to some practitioners and they say it is difficult to get the owners to come back for these repeated injections, but we do not find it so. We make a note on the calendar, when the next dose of vaccine is due and send a note to the owners and they come back. We explain to them, with the initial injection, that it means an expenditure of $20.00 during the first two years of the dog's life. Being spread over such a period of time, it is easy to pay and constitutes inexpensive insurance against distemper. We charge five dollars for injecting vaccine alone. For the initial dose of serum, given with the first dose of vaccine, we do not make an extra charge.

DR. J. M. ARBURUA: I would like to ask Dr. Longley this question: When an animal comes in to be hospitalized, you say you give them the serum and the vaccine. Do you do that on the same day?

DR. LONGLEY: Yes.

DR. ARBURUA: You don't give the serum one day and follow two or three days later with the vaccine?

DR. LONGLEY: No, unless we suspect that the dog has been exposed to distemper. Then he gets the serum alone, and the vaccine is not given unless he is going to remain in the hospital for more than a week. We figure the serum will carry him a week or ten days, but not longer. Supposing he should not pick up any distemper virus until six, seven or eight days after receiving the serum. The period of incubation is long enough so that the serum will have completely disappeared from his system by the time the distemper virus gets in its work. If a dog remains in the hospital for two weeks, and there is any reason to suspect that we should not chance giving the vaccine, on account of the possible exposure, we give another dose of serum. Take the spaying cases. The initial dose of serum will carry the dog for a week, but if the dog remains two or three days over a week, then we give another injection of serum alone.

DR. ARBURUA: I would like to ask Dr. Longley another question: You suggest giving the vaccine at two months, four months and six months. The animal, when you are through vaccinating him, is a year old. Is that not so?

DR. LONGLEY: Let us do a little figuring. The animal, on the average, is three months old at the first injection; two months more makes him five months old; four months more makes him nine months old; six months more makes him fifteen months old.

DR. ARBURUA: You say that will make him immune for life?

DR. LONGLEY: The final injection should carry immunity longer than the initial injection because he is an older dog and probably has acquired immunity by exposure before he gets the last injection.

DR. ARBURUA: We have had the preparation only a year. I was just wondering how you worked it out?

DR. LONGLEY: Dr. Wood, when did you first come out with the vaccine? More than a year ago?

DR. WOOD: Yes.

DR. EHMER: I would like to ask Dr. Longley a question: The spaying case, or whatnot, that we are going to keep for a week, are you going to allow the dog to go out where other dogs are or keep it in a cage by itself?

DR. LONGLEY: We take every possible precaution to prevent undue exposure. We have separate rooms and the rooms where the surgical cases are kept are removed from the distemper cases as much as it is possible to arrange it. However, in these surgical wards, even though they are removed from the distemper cases as much as it is possible to remove them and every precaution was taken, we had breaks from those rooms prior to the advent of modern immunizing agents.

I might describe that further by saying that before the vaccine came out we put up a brand new country boarding kennel. We were extremely careful not to take in any dogs that showed evidence of distemper; we didn't take in any sick cases for treatment. The first year distemper broke out among the young dogs, and they had no known exposure, showing it cannot be done under any condition, without following some form of immunization. We find the same breaks in a new kennel, with each dog in individual quarters, as we do in an old place where we know it is infected. The theory with me is that there are mature dogs that are carriers of distemper virus.

Another point just came to my mind: The question has been asked whether or not the injection of vaccine, or, as Dr. Wood calls it, antigen, which is a dead, or nearly dead, virus, at the time of production, has any unfavorable influence. It has not been our experience that it has. We can give a dog serum and vaccine and spay her the same day and have her go home with no unfavorable reaction.

I concur with the doctor's suggestion that by making the injection, if it is done with one insertion of the needle, over as large a subdermal area as possible, you won't have any trouble with abscesses. It is leaving it in a pocket that causes the abscesses.

DR. J. J. HOGARTY: I have two questions that I would like to ask. First, is there any time in the natural life of an unvaccinated dog that he is immune to distemper infection? Second, does an attack of distemper confer permanent immunity?

The object in asking the first question is that frequently people come to me and say, "My dog is too old to contract distemper. My doctor tells me that if he is over a year old he is immune and he won't take distemper." I would like to say I think they are in error in giving such information to their clients. However, I would like to hear the opinion of some of the men who have had experience along that line.

DR. LONGLEY: I think that question can best be answered by emphasizing the point that immunity is not a definite something; it is relative and the only statement that can be made as to whether a dog is immune at any particular age, or one attack confers immunity for life, is to take the average case. It doesn't make any difference what the rule is in immunity, there is an exception occasionally. A dog may be highly immune to distemper today and a month from now something happens, cold, exposure, hunger, intestinal parasitic infestation—a thousand things can happen to lower his resistance and his immunity will disappear. We find that in all animals. Cattle are highly immune to blackleg after having been properly vaccinated. If driven over a long distance, tired out, and no chance given them to eat, and they are put on infected pastures, you will get a break. The principle is the same in hog cholera.

Some dogs seem to be naturally immune to distemper and never do take it. If they do, they have it in such a mild form that it is not recognized. But I believe that in the vast majority of cases, either they have been exposed to, or have acquired immunity to, distemper before they are two years old, or they have had an attack and recovered by that time. I don't know how you are going to figure it out—but it is so close to 100 per cent that the exceptions are extremely rare. I have seen only a few cases where a dog over two years old actually took distemper. I have seen some two-year-old German shepherds taken down, but few older than that. I have seen dogs twelve years old going through symptoms that appeared to be distemper, and I would diagnose the cases as distemper, but I haven't seen many of them in my experience.

DR. HOGARTY: My point is this: Is it proper for a veterinarian to give a client the information that his dog is immune from distemper after two years of age? It isn't a question of understanding immunity. I know about the blackleg, etc. But is it proper for a veterinarian to say that if your dog is two years old he is immune to distemper? I don't think he is.

DR. LONGLEY: That may not be exactly proper. But it seems proper to me that that is a proper statement to make, with the added exception that occasionally a case develops after that, but it is so rare it doesn't need to be considered. That is what I say. It leaves you an out, at least, by adding that sentence.

DR. J. F. MCKENNA: I am glad Dr. Hogarty brought this question up, because I think it is one we meet with in practice every day. Following the work done by Lansing, about 1915 or 1917, in which he experimented with hundreds of cases, in my practice I have carried it on over a number of years and I agree with Dr. Longley. I think it is the exception and not the rule, where a dog over two years old comes down with true canine distemper. I think it is the experience that they are secondary infections, and not true canine distemper, and I think it is safe for the veterinary profession, not to tell their clients 100 per cent but a differentiation in diagnosis should be made between the young dog with distemper and an old dog with symptoms which resemble distemper.

We have exposed hundreds of dogs to canine distemper, both in Fresno and in Los Angeles. In Fresno we have deliberately exposed pound dogs and stray dogs in the distemper ward and seldom, or never, have we been able to produce in an old dog true canine distemper such as affects young puppies. We have been able to take these same dogs and expose them to the infection which occurs in four-, five- or six-year-old dogs, and they will show symptoms of distemper, but I doubt whether that infection is true canine distemper.

DR. J. C. FLYNN: The question that Dr. Hogarty asked, I think is a very good question. I have had twenty years of experience in handling those kinds of cases and I find that dogs are never immune to distemper until they have

had a case of distemper—I am speaking of unvaccinated dogs—and my experience has been that every dog that has had distemper is absolutely immune throughout his life. In support of that statement I am going to recite a few cases.

I have, at the present time, one two-year-old setter, a male dog, that had distemper when he was about two months old. He has been in the hospital about two years. I have another one that is nine years old and that had distemper when it was six months of age. I have a pointer—these are my own dogs—that had distemper when he was just about two years of age. Those dogs have been in the hospital practically all of their lives. Whenever I desire to do so, I take those dogs, because they are my property, and I put them in my contagious ward, if I am crowded in other wards. They go in there and when they come out they are given antiseptic baths and put back into the other run. Those dogs have never shown any indications of coming down with distemper or showing any evidence of distemper.

At one time I had a young Boston terrier brought to me at the age of about six weeks. He was badly infested with intestinal parasites. He was cleaned up from that condition and he contracted distemper in the hospital. I nursed him through distemper and took care of him and that dog was in the hospital as a patient almost his entire life of eight years, when he was taken out by the owner and destroyed. That dog had a little weakness of the stomach, a little gastritis, that troubled him, when not in our care. He never was sick in the hospital. Every time they took him home and gave him different food, he would vomit on the carpet and back to the hospital he would be sent. He was there practically eight years. He was in the contagious ward any time we wanted to put him in there. After the first attack of distemper he never showed symptoms again.

I recall one instance, speaking of the age at which animals may contract distemper, a little black and tan that was fourteen years of age. It was a house pet and had been kept in the house all of its life. It had a little eye trouble. It was sent to a hospital and contracted distemper in that hospital. It was brought home and had a typical case of distemper. Some ten days after it had been brought home, a young dog in the same house came down with distemper, which proves that the dog fourteen years of age contracted distemper and carried distemper back to the home. That little black and tan died with one of the most virulent cases of distemper I ever saw.

I firmly believe that one attack of distemper gives or confers permanent immunity. I believe that until a dog has distemper he is not immune, regardless of age.

DR. J. B. HARRISON: I would like to take exception to part of Dr. Flynn's statement. I think that the pups that get distemper, and you catch it in the first rise in temperature and treat them with homologous serum, have had distemper, but in too mild a form, and they will get it at some later day, say three or four months later, and it will be true distemper in both cases. But if they go through distemper and have the secondary invaders there, that gives them their permanent immunity. But if you catch a case of distemper in the primary stages and stop it with serum, which can be done, with young pups it doesn't confer life-long immunity.

DR. LONGLEY: May I ask the doctor on my right if he has had enough cases down with distemper, stopped in the initial stages with homologous serum, to prove that statement?

DR. HARRISON: Yes sir.

DR. LONGLEY: We have had the opposite experience. We have had lots of those cases that remained in the hospital through their entire lives, up to the present time, and have been repeatedly exposed.

DR. FRICK: I would like to stress just one point that Dr. Wood brought out, I think you are overlooking it.

I happen to have close connection with the racing greyhound game in this country. We have a condition with them that is not well understood. We have two in our hospital now—Bo McMillan and Golden Bubbles—two dogs that were shipped up from Florida. Those dogs came in—they had been on the track—to the hospital not feeling well. They had diarrhea and there was pus in their eyes. Those dogs did not have distemper. The veterinarian who

sent them to the hospital said they had distemper. We have a number of veterinarians in different sections of the country diagnosing these cases as distemper, dogs that have been on the track for eight years. Bo McMillan is eight years old. It looks like distemper, but it is not. He has been in the hospital for three months with infectious enteritis, common on the tracks down in Tia Juana and Florida and around St. Louis. That is not distemper. And yet I can name four or five veterinarians in this country who diagnose it as distemper.

You put those dogs in with susceptible dogs and the susceptible dogs, puppies, do not contract distemper. Later on they can pick up distemper. That condition is not distemper and I do not think it is very easy to diagnose distemper in many of these cases.

DR. A. A. HERMANN: The statement was made, I believe, that dogs, puppies, with immune mothers, are not susceptible to and do not contract distemper until after weaning time. I do not claim to know much about distemper. I have had as much grief with it and perhaps as extensive experience with it as most men, and it is my experience that puppies kept in infected quarters will shrivel up and die with an infection frequently before they are two weeks old when their diet is nothing but the matron's milk. In puppies that seem to be developing this disease, which evidently is contagious, since it spreads rapidly from one pup in the litter to additional pups, if given a dose of homologous serum, which we almost consistently give within twenty-four hours after their birth, and in appropriate doses, favoring about 1 cc for about each four ounces of weight, we find that we can prevent the development of this illness. Since homologous serum is the only thing given, it must have some relationship and some bearing on the situation.

DR. O. B. MORGAN: I would like to ask Dr. Frick if he makes a microscopic examination for coccidiosis in the greyhounds. We have observed some clinical symptoms of coccidiosis in dogs.

DR. FRICK: Every dog that comes in gets an examination, no matter what he comes in for. We have lots of students and we always have an opportunity to make examinations. Coccidiosis seems to be on the increase, but we have not been able to find that condition in Kansas, even in the dogs shipped up from Texas and the South.

DR. LONGLEY: Just a further word in regard to the immunity of pups with nursing mothers. I guess I didn't make my statement quite clear. In our hospital we have fifteen brood bitches of the small breeds under conditions identical with surgical cases and boarders. These pups that appear to be immune from the mother's milk are carried in exactly the same environment that the mother is. If an outside brood bitch were brought into the hospital, I would consider some form of immunization necessary. What I mean is this: That if there is any possible chance for those pups to pick up virus infection, the mother is highly immune because she also is repeatedly exposed. That would not apply to a mother or a brood bitch brought in from the outside.

DR. WOOD: In regard to Dr. Steel's question, I think he has made some criticism of commercial houses for not giving complete enough information on products. Well, that criticism may be in order, but I am always glad to give Dr. Steel any information I can. I merely wish to say that a commercial house is not an experimental laboratory.

On the antigen, deterioration does occur and the antigen should be handled carefully and in the same way that other perishable products are handled. One can trace antigen all the way from virus to the point where there is no immunizing response from its use at all. I use the term antigen or vaccine. These titles indicate the same product licensed by the Bureau under two names. Deterioration of virus is not regular. Some lots die off promptly, while others may be maintained for considerable periods under proper storage conditions.

I talked this morning with probably a little bit of pessimism, but what I wanted to do was to point out some of the facts regarding this immunization against distemper that apparently were overlooked by many following the publication of the original work of Laidlaw and Dunkin. Nearly everyone accepted this report with a great deal of enthusiasm, but did not bother to read carefully between the lines.

The potency of serum depends entirely upon the condition of the serum dog at the time of hyperimmunization and on the dosage and virulence of the virus given. Virus is better for serum-production purposes when obtained from virus dogs early in the disease. The virus is present in the blood-stream and in the organs, but early virus will produce a more effective serum that virus obtained during the later stages of the disease. This does not follow with vaccine or antigen. Here your tissues produce better antigens for immunizing purposes when selected later in the disease.

The potency of serum can be tested just as accurately as the potency of serum for hog cholera, and in the same way. The only trouble is obtaining a good source of serum-test dogs. There is great variation in dogs as to their susceptibility to distemper and one might be badly fooled on a test if the test dogs were not sufficiently susceptible to distemper.

I think that at present you will all agree with me that the most hopeful product for the immunization of dogs against distemper—I am talking about the present—is in an improved antigen, or vaccine, since this product is safe to give and there is no danger of producing infection with it.

As to the method of vaccination, the practical method is to give serum, to protect against previous infection, that is, infection just prior to vaccination or infection just after vaccination; with antigen followed by a second dose of antigen. I would say virus, if one could depend on virus, but I don't think we can depend on it at present.

In regard to hospitalization, you men as practitioners have had more experience with this problem than I have had, but my opinion would be that one should depend on serum alone, since the vitality of animals undergoing surgical operations is lowered and there is some lowering of body resistance for a few days after giving the antigen or vaccine, which would further depress resistance and it is for these reasons that I would think serum alone should be depended upon. One can, of course, neutralize the negative phase following vaccine by the use of serum. But I would favor serum alone for the ordinary surgical case.

The dosage of vaccine can be varied. A 5-cc dose is not too large for the small dog; and one will, of course, get a greater immunizing response with a larger dose. Therefore, it is good practice to increase the dosage of antigen or vaccine when immunizing large dogs.

Age and immunity: Old dogs are just as susceptible to distemper as young dogs providing they have not been in contact with distemper. There may be exceptions but they are rare.

Here is a point that I haven't heard anyone mention—mild cases of distemper—probably the animals which are responsible for most of the spread of distemper. One may have cases of distemper so mild that a diagnosis is very difficult, and these cases will carry distemper as effectively as a very sick dog. The symptoms of distemper are very often obscure, but personally I would make a diagnosis on fever, inappetence, rapid wasting and evidence of infectiousness. Coughing, pneumonia or enteritis are not necessarily symptoms of distemper.

Carriers: I do not believe they exist. When a dog has made a complete recovery from distemper I do not think there is any danger. This is a hard point to prove but I do not think we will find carriers.

There is one other possibility to be considered in discussing distemper. That is the possibility of the existence of more than one virus. This has never been shown, but should be kept in mind.

Dr. Steel: I would like to explain my position to Dr. Wood. It was not my intention to be critical at all, but I wanted to make the statement that the manufacture of homologous serum is not standardized. In the production of anti-hog cholera serum there is a definite plan that you cannot get away from and that is outlined by the Government. There is no standard plan for the production of homologous serum made in this country, as far as I know. Furthermore, there are no tests that are required by the Government. I am in intimate touch with the veterinarian in charge of virus inspection work in Kansas City and I know this to be true, because I have gone over that with him recently. If there is a possibility of standardizing serum-production methods, I think the practitioners are entitled to have some consideration along that line from the Government.

We are using the term "homologous serum." As I say, it may be homologous serum and not homologous distemper serum. You cannot use that term indiscriminately. I think that as far as we know, the giving of homologous serum is an additional protection and, as Dr. Wood pointed out, there are variations. Insofar as the method is not standardized, we may not be conferring a lot of immunity; then again we may, by the giving of homologous serum. We cannot give it and then put the dog down in the distemper ward with impunity.

DR. WOOD: In regard to the production of homologous anti-canine distemper serum, Dr. Steel's statement is exactly right. There is no test required by the Bureau. However, producing houses must supply the Bureau with their method of production and it must meet with the Bureau's approval. I think the reason for that is this: The product is new and there is no recognized standard procedure at present. It is one of those things that will come, but I think that is the reason.

BUREAU TRANSFERS

Dr. Clifton Carter (K. C. V. C. '08) from Amarillo, Texas, to Reno, Nevada on field investigation.

Dr. Asa Winter (Mich. '21) from Lansing, Mich., to Albany, N. Y., on tuberculosis eradication (stationed at Oswego, N. Y.).

Dr. B. C. Johnson (U. S. C. V. S. '20) from West Fargo, N. Dak.. to Albert Lea, Minn., in charge of meat inspection.

Dr. W. L. Carson (Chi. '13) from Missoula, Mont., to Great Falls, Mont.

Dr. James E. Ewers (K. C. V. C. '10) from Pheonix, Ariz., to Sacramento, Calif.

Dr. Otto Hornlein (McK. '10) from Austin, Minn., to Indianapolis, Ind., on meat inspection.

Dr. Byron N. Lauderdale (A. P. I. '17) from Saint Augustine, Fla., to Orlando, Fla., on tick eradication.

Dr. John L. Myers (Colo. '24) from South Saint Paul, Minn., to Menominee, Mich., on meat inspection.

Dr. E. M. Nighbert (Ont. '94) from Moultrie, Ga., to Washington, D. C., on extension work in parasite control with the Zoological Division.

Dr. T. A. Shipley (Chi. '90) from Denver, Colo., on meat inspection, to Council Bluffs, Iowa, on hog cholera control.

Dr. Henry Tornow (K. C. V. C. '09) from East Saint Louis, Ill., to Zionsville, Ind., on virus-serum control.

Dr. J. R. Aufente (U. S. C. V. S. '06) from Honolulu, Hawaii, to Spokane, Wash., on meat inspection.

Dr. Francis S. Bingham (K. C. V. C. '07) from Topeka, Kans., to Providence, R. I.

Dr. Norman G. Cobbett (Colo. '21) from Galesburg, Ill., to Lamar, Colo.

Dr. W. J. Conlin (Corn. '20) from South Saint Paul, Minn., to Milwaukee, Wis., on meat inspection.

Dr. M. L. Crans (K. C. V. C. '16) from Philadelphia, Pa., to Terre Haute, Ind., on meat inspection.

Dr. C. H. Deisel (Cin. '11) from Logansport, Ind., to Chicago, Ill., on meat inspection.

Dr. R. S. Griffin (Chi. '08) from Salt Lake City, Utah, to Walla Walla, Wash., on meat inspection.

Dr. Hilborn Groat (K. S. A. C. '21) from Chicago, Ill., to Topeka, Kans., on meat inspection.

Dr. Elils E. McCoy (K. C. V. C. '07) from Charlottesville, Va., to Trenton, N. J., in charge of tuberculosis eradication.

Dr. O. W. Noyes (K. C. V. C. '04) from Kansas City, Mo., to Evansville, Ind., on meat inspection.

Dr. Ray O. Porter (K. C. V. C. '09) from Kearney, Nebr., to Edmond, Okla., on tuberculosis eradication.

A NOTE ON AVIAN LARYNGOTRACHEITIS*

By ROBERT GRAHAM, FRANK THORP, JR., *and* W. A. JAMES,
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For many years the occurrence of avian laryngotracheitis, erroneously known as infectious bronchitis, has presented a problem of increasing significance to flock-owners, hatcherymen, poultry-feeders and shippers. Aside from the death losses traceable to this and clinically related maladies, the short feeding period adopted at concentration points in order to prevent outbreaks of the disease in mature fowls has noticeably interfered with poultry-feeding operations in the Corn Belt. In farm flocks hasty marketing has been employed to avoid losses, and fowls in the incubation stages of the disease have frequently arrived at market centers showing pronounced symptoms. Though the principal losses have been sustained in mature fowls, the occurrence of a clinically related respiratory infection in battery brooder chicks or broilers has in recent years been reported in many localities throughout the Mississippi Valley region. The losses from clinical laryngotracheitis in young chicks include not only a significant death rate, but limited gains or delayed growth and development in chicks that survive the infection.

Aside from the acute type of the malady, a subacute laryngotracheitis in mature fowls and young chicks, not clinically resembling the acute malady, also has been observed. Fortunately, in our experience, the subacute syndrome is accompanied by a low mortality in mature fowls. Observations in Illinois flocks suggest the occurrence of two clinically distinct types of laryngotracheitis with variable symptoms. There is also the possibility of distinct and separate infections characterized by analogous symptoms as well as the possibility of a virus or viruses producing a varied clinical picture of infection dependent upon environmental factors and the age of the host. The former seems more tenable in view of our observations.

BACTERIOLOGIC STUDIES

Since 1923, specimens of natural laryngotracheitis in fowls of different ages have been examined bacteriologically and path-

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ologically at the Illinois Agricultural Experiment Station, while laryngeal exudates from naturally infected fowls have been repeatedly employed to expose healthy fowls. The results of these investigations[1] suggest that more than one clinical type of the disease exists and that the so-called bronchitis syndrome in fowls is primarily a laryngotracheitis, while the course of the infection or infections observed in naturally infected flocks may be either acute or subacute and chronic in character. Associated with this respiratory disease or group of diseases, a toxemic syndrome of varying severity also has been observed. In such cases primary gross lesions of laryngotracheitis may not be grossly detectable. Chicken-pox virus in the respiratory tract, as reported by Gwatkin,[2] has been encountered in only a few outbreaks, yet the prevailing acute and subacute avian laryngotracheitis in Illinois flocks encountered in our investigations is not associated with gross lesions of chicken-pox, while the laryngeal exudates of naturally infected fowls have repeatedly proven negative to this or other demonstrable filtrable viruses.

A variety of non-pathogenic or semi-pathogenic microörganisms have been encountered in the laryngeal exudates of acutely and subacutely infected fowls, though the internal organs of naturally infected mature fowls are relatively free from bacterial invasion. Among the microörganisms encountered in the laryngeal exudate of naturally infected mature fowls and young chicks was a pleomorphic type with coccoid, diplococcoid, and barred rod forms possessing some of the characteristics ascribed to the diphtheroid group. In a study of these and other semi-pathogenic microörganisms isolated from spontaneous cases of the disease, the diphtheroid-like types encountered displayed some invasive properties for the upper respiratory tract of healthy fowls, and attention was given to this group to note their frequency in natural or spontaneous outbreaks of laryngotracheitis. Healthy chicks and mature fowls without a history of respiratory diseases have proven relatively free from the types referred to in this discussion. The presence of pathogenic diphtheroid-like microörganisms in the laryngotracheal exudate of naturally infected fowls therefore prompted a series of experiments to determine their relation, if any, to an acute and subacute spontaneous avian laryngotracheitis occurring in young chicks and mature fowls.

Artificially Induced Laryngotracheitis

In human pathology, diphtheroid types have been encountered so frequently in a variety of pathological conditions that little or no significance is attached to their presence. In this connection, it may be well to mention that different diphtheroid microörganisms encountered in laryngotracheitis of fowls have also proven non-pathogenic and the report presented herewith is not intended as evidence of the pathogenicity of the general diphtheroid group but to call attention to certain pathogenic diphtheroid-like types encountered in avian laryngotracheitis. Diphtheroid types described by Mellon,[3,4] Hektoen,[5] and others in man are easily differentiated from the pathogenic avian diphtheroid-like types on the basis of beta hemolysis, yet this character has been noted in certain ovine diphtheroid strains described by Marsh.[6]

In our experiments, a variety of microörganisms encountered in the laryngeal exudates of naturally infected fowls have been injected in healthy chickens of different ages. The pleomorphic avian diphtheroid-like types proved capable of inducing a subacute laryngotracheitis following postglossal or intralaryngeal instillation, while intravenous inoculation of healthy chickens of different ages produced an acute edema and congestion of the larynx. Larger doses intravenously injected frequently produced a profound toxemia and death in fowls of different ages. Accumulations of mucous or fresh bloody extravasations in the larynx and upper trachea, as observed in natural acute cases of laryngotracheitis, may be artificially reproduced occasionally by this method of exposure, using a single strain of the pathogenic avian diphtheroid-like microörganism. Likewise, subcutaneous inoculation of young chicks (4 to 10 weeks of age) with certain diphtheroid-like types, isolated from acutely or subacutely infected fowls, may produce a toxemic syndrome and often death. Symptoms of depression which follow subcutaneous injection of young chicks resemble the acute toxemic laryngotracheitis. The symptoms appear in 6 to 8 hours or longer after subcutaneous exposure, emphasizing the toxemic rather than infective character of the virus, while death may take place in 1 to 5 days of young chicks following this type of exposure. The syndrome induced may or may not be associated with mouth breathing, as noticed in the natural acute respiratory infection, but in no case have the acute respiratory manifestations appeared following exposure to avian diphtheroids. Often times, the natural acute respiratory symptoms suggest cardiac insufficiency rather than uncomplicated

laryngeal stenosis terminating in death from suffocation, while histopathologic changes in the myocardium, kidneys, and liver of natural cases, as well as the diphtheroid-induced disease, show numerous hemorrhages throughout the endothelial cell structures.

Aside from the experimentally demonstrated invasive character of different diphtheroid-like types isolated from natural acute cases of the disease, the clinical as well as bacteriological findings with reference to pathogenic diphtheroid-like types in the upper respiratory tract of subacutely infected fowls suggest the possible carrier feature of this pathogen of low-grade virulence in mildly infected fowls or the occurrence of a spontaneous, non-fatal, subacute or chronic form of a diphtheroid infection, which may be so mild in mature fowls as to escape attention or fail to impress the owner of the existence of a specific disease.[7] Moreover, the subacute type of avian laryngotracheitis, as judged by clinical deductions and gross lesions in the larynx of naturally infected fowls, and by repeated direct passage of a subacute laryngotracheitis infection or infections by laryngeal exudates of natural acutely infected fowls or fowls artificially infected with the avian diphtheroid-like microörganisms, does not clinically resemble the naturally acute laryngotracheitis accompanied by respiratory symptoms. Therefore, there is the possibility of separate and distinct etiologic infections in the acute and subacute disease, not excluding filtrable agents which, in keeping with the character of known filtrable virus diseases of the respiratory tract, might disappear after the initial symptoms or be demonstrated with difficulty in the exudate of the respiratory tract at the peak of the disease. In fact the results in attempting to communicate an acute natural laryngotracheitis accompanied by respiratory distress suggests that certain agents present in the natural acute disease are not successfully propagated by laryngeal transfers beyond one or two generations or communicated by contact exposure consistently, yet it appears that a subacute laryngotracheitis encountered in our observations may prevail separate from an acute form of the disease. This phenomenon has been repeatedly noted in the course of the natural disease in farm flocks. The possible cyclogenic character of a laryngotracheitis virus or viruses is also suggested in the subacute termination of the natural acute laryngotracheitis. The natural subacute infection, like the artificially induced diphtheroid laryngotracheitis, may be communicated to healthy fowls by direct laryngeal

swabs and by contact, but the mild character of the malady in mature fowls does not resemble the acute laryngotracheitis.

The natural or environmental factors which may influence the virus or viruses in the development of the acute type of laryngotracheitis appear difficult to reproduce under laboratory conditions, even though the unaltered laryngeal exudates are employed in propagating the disease. For the reason that it has proved impossible to propagate the acute disease continuously by direct laryngeal passage, it appears that the acute and subacute types of avian laryngotracheitis in Illinois may be due to different causes or inseparably associated with a virus or combination of viruses that are not easily propagated under artificial conditions. This conjecture may explain the difficulty experienced in consistently duplicating the acute malady under laboratory conditions, while its culmination in a clinically subacute laryngotracheitis as well as the gross lesions observed resembles the artificially induced subacute diphtheroid-like infection. The possible presence of two etiologic agents not unlike the canine distemper etiology is not excluded in our observations.

The natural subacute type of avian laryngotracheitis in contrast to the acute may be repeatedly transmitted from fowl to fowl as judged by symptoms and lesions, by placing the laryngeal exudate of naturally subacutely infected fowls into the larynx of healthy fowls. The culturally induced inflammatory lesions may likewise be repeatedly communicated from fowl to fowl by direct swabs. Experimental efforts to stimulate the development of the acute disease in subacutely infected fowls (natural or from cultures) have proven negative, notwithstanding observations in the field which suggest such a transformation as well as the possible invasion of an independent virus becoming superimposed on the subacute disease.

Single strains of the avian pathogenic diphtheroid-like microörganism following instillation into the larynx do not produce the clinical acute type of avian laryngotracheitis observed in mature fowls, yet chicks two to four weeks of age may frequently succumb from a toxemia following intralaryngeal instillation of cultures. The clinical syndrome induced in chicks resembles the natural toxemic disease. Freshly isolated diphtheroid-like microörganisms apparently possess greater invasive properties than strains artificially propagated over a long period of time, yet a variation in the invasive properties of different strains has been repeatedly noted.[8] Mature fowls exposed via the larynx may

show transitory laryngeal inflammatory processes of varying intensity, accompanied by intermittent shaking of the head, repeated swallowing, and wheezing respiratory sounds.

Cultural Characters

The pathogenic avian diphtheroid-like microörganisms isolated from natural outbreaks of the disease possess similar cultural and tinctorial characters. Pleomorphism on different media is noticeable while fixed morphological types on the same media are not constant. Coccoid, diplococcoid, ovoid, and barred rod types have been observed. They are non-motile, Gram-positive, facultative anaerobic, and do not form spores. Slight variations in carbohydrate fermentation have been noted. Fermentation occurs in mannitol, galactose, maltose and sucrose without gas production, while in lactose, dextrose, raffinose, inulin, salicin, levulose from inulin, mannite, rhamnose, xylose and arabinose, acid production is slight or inconsistent without gas formation. Chicken-infusion-broth agar containing 10 per cent horse blood has been most successfully employed in the isolation and propagation of the pathogenic diphtheroid-like microörganisms encountered in avian laryngotracheitis. Growth on plain chicken-infusion-broth agar is inconsistent or negative.

Serologic Types

At least three types of pathogenic avian diphtheroid-like microörganisms have been recognized in agglutination tests (table I), with specific agglutinating sera prepared in horses and rabbits by repeated subcutaneous injections of dead and living cultures.[9] More than one serologic type of diphtheroid-like microörganisms has been encountered in the laryngotracheal exudate of fowls in a single outbreak. Low dilutions may not

Table I—*Serologic types of avian diphtheroid-like microörganisms*

Antigen	Type 1 Serum					Type 2 Serum					Type 3 Serum				
	400	800	1600	3200	C	400	800	1600	3200	C	400	800	1600	3200	C
Type 1	+	+	+	+	−	−	−	−	−	−	−	−	−	−	−
Type 2	−	−	−	−	−	+	+	+	+	−	−	−	−	−	−
Type 3	−	−	−	−	−	−	−	−	−	−	+	+	+	+	−

C=Control

consistently differentiate the serologic types, though a definite demarcation is invariably observed in dilutions of 1:400 or above. The existence of even more than three specific types is suggested on a serologic basis as agglutination does not occur with some strains isolated.

Laryngotracheitis in Young Chicks

The clinical laryngotracheitis observed in mature fowls or a closely allied infection has been observed in chicks of different ages. As a rule the toxic syndrome in chicks is more common than in mature fowls, yet an agonal dyspneic syndrome of short duration may often be observed. The toxic syndrome in young chicks may be complicated with pullorum disease yet evidence incriminating the latter as the causative factor of laryngotracheitis has not been substantiated in our studies. The disease in young chicks is marked by weakness, depression and cardiac arrest, with only slight laryngeal inflammatory changes. In some flocks no symptoms have been observed though a number of chicks were found dead. At autopsy of naturally infected chicks the gross lesions may, however, appear comparable to those in mature fowls, though the local edematous, mucous, fibrinous, or exudative inflammation of the larynx is not so commonly noted as a congestion of the lungs, liver and kidneys. Whitish exudative patches are occasionally noted in the laryngeal and postglossal regions. Microscopically hemorrhagic lesions are pronounced in the lungs and heart and are in accord with the toxemic type of the disease which may characterize the infection in mature fowls.

From the respiratory tract of naturally infected chicks, pathogenic, pleomorphic, diphtheroid-like microorganisms, culturally and serologically indistinguishable from similar types encountered in the respiratory tract of naturally acutely and subacutely infected mature fowls, have been isolated. The pathogenic character of a few strains isolated from baby chicks, as judged by symptoms and lesions induced in healthy chicks following instillation of cultures into the larynx, has been demonstrated. Subcutaneous injection of cultures produces laryngeal inflammatory changes in young chicks but mature fowls appeared more resistant to this type of exposure.

Summary

1. So-called avian infectious bronchitis appears to be an infectious laryngotracheitis. The acute clinical disease observed in

our studies is communicable to healthy fowls in initial contacts, but has been successfully propagated by this method of exposure only temporarily. Similar results were experienced with laryngeal swabs. On the other hand a subacute type of laryngotracheitis may be propagated by this method. Thus the experimental as well as epidemiological evidence suggests that more than one etiological and clinical type of the disease prevails.

2. Diphtheroid-like microörganisms isolated from the laryngotracheal exudates of naturally acutely and subacutely infected fowls of different ages appear incapable of producing an acute laryngotracheitis in healthy fowls following laryngeal instillation. However, certain diphtheroid-like strains appear capable of producing a subacute inflammation of the larynx and trachea. In young chicks subcutaneous injection as well as intralaryngeal instillation may prove fatal.

3. In the subacute form of laryngotracheitis in mature fowls, respiratory distress is often mild and momentary in character. It is nonfatal but may be communicated by contact or laryngeal swab. The acute form of laryngotracheitis in mature fowls is characterized by difficult respiration and symptoms of profound toxemia. In young chicks the toxemic complex appears to be more frequent than in mature fowls. Laryngotracheitis in young chicks may occur independently or in association with pullorum disease.

4. Chicken-pox virus has been encountered in relatively few outbreaks of avian laryngotracheitis coming to our attention. This virus is capable of causing acute respiratory distress following laryngeal instillation but in our observations is not responsible for the prevailing acute or subacute laryngotracheitis in Illinois flocks.

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The Epizooty

Under the head, "Fifty Years Ago," the Raleigh (N. C.) *Times* recently reproduced the following interesting item:

October, 1880

The epizooty is approaching. Several horses are thought to have it now. It is very severe in the North.

CHLOROFORM-TREATED RABIES VACCINE*

By MAJOR R. A. KELSER, V. C.,

Army Medical School, Washington, D. C.

In September, 1928, the writer published a preliminary note[1] on the use of a chloroform-treated vaccine for rabies immunization. Since that time this work has been continued in our laboratory with results confirmatory of our earlier conclusion that such vaccine was superior to a number of the products heretofore commonly used for rabies immunization.

Because of the lack of facilities for maintaining dogs at the Army Medical Center, our potency tests of this vaccine have been carried out with rabbits. However, vaccine prepared according to our method has been subjected to comparative potency tests with other vaccines, on dogs, by Schoening, of the United States Department of Agriculture, and his results[2] have been in line with those we have obtained with rabbits.

We have recently vaccinated with this vaccine all of the dogs (approximately 150) at one of the Army posts, and arrangements have been made to repeat the vaccination of these animals annually for a period of five years or more. The incidence of rabies among the animals at this post will then be compared with the incidence of the disease in dogs belonging to civilians in the city in which this post is located.

When our work with rabies vaccine was first inaugurated, we were of the opinion that the chloroform rendered the rabies virus inactive within a very short time, as it does rinderpest virus. This conclusion was based on the fact that rabbits inoculated subdurally with our initial lot of vaccine, prepared from a Philippine strain of fixed virus, failed to develop rabies, and a number of animals subsequently given subcutaneous injections of this and other lots of vaccine showed no ill effects as a result thereof. However, after resuming our experiments at the Army Medical School, following the return of the writer to the United States from the Philippine Islands, we had one rabbit, in a group which had received a two-day-old vaccine subcutaneously, develop rabies thirteen days later as a result of the vaccine injected. This immediately led to a series of experiments which

*Presented at the sixty-seventh annual meeting of the American Veterinary Medical Association, Los Angeles, Calif., August 26-29, 1930.

indicated that with some lots of vaccine the chloroform was slower in definitely inactivating the virus than we had at first assumed. As a result of a number of tests wherein vaccine of different ages was administered subdurally to rabbits, we have found that the product should be aged at least two weeks to insure that the virus has been definitely inactivated. Further, in connection with the inactivation of the virus in this type of vaccine we feel that two factors are very important. One is a uniformly very finely divided tissue content. The other is the shaking process, wherein the vaccine is well shaken two or three times a day during the two weeks it is permitted to age.

Preparation of the Vaccine

In our earlier work the chloroform content of the vaccine was 0.75 per cent. This has been subsequently increased to 1 per cent. The vaccine we are now using is prepared in the following manner: Rabbits weighing approximately 2½ kilograms are inoculated subdurally with fixed virus. After developing marked symptoms of rabies (general paralysis) they are bled to death and the brains carefully removed with aseptic precautions. This tissue is them weighed in a sterile, tared container and then triturated into a smooth paste in a sterile mortar. Then, while still triturating the mass, there are slowly added 2 cubic centimeters of sterile physiological saline solution for each gram of tissue. This emulsion is then worked through a sterile 40-mesh wire sieve to insure that the tissue is very finely divided. Having the tissue free of coarse particles obviously insures against clogging syringe needles, but further, as already indicated, facilitates the action of the chloroform on the virus. The mixture is placed in sterile stock bottles and 1 per cent chloroform (c.p.) added and the bottle tightly stoppered, well shaken and stored in the refrigerator where it is permitted to age.

In placing the vaccine in the stock bottles we use a sterile, long-stem funnel and endeavor not to contaminate the inside of the neck of the bottle with vaccine. Then, as an added precaution, we thoroughly flame the neck of the bottle to insure that there will be no vaccine containing live virus between the neck and the stopper.

During the period of ageing, the stock bottles containing the vaccine are well shaken for approximately 5 minutes 3 times a day.

In our work with rabbits the dose of vaccine has been 2 cc for rabbits weighing approximately 2½ kilograms. In the beginning we administered two doses of vaccine with an interval of one week between the doses. Two weeks subsequent to the last dose of vaccine the rabbits were infected subdurally with 0.10 cc of a 1:100 dilution of street virus. Of late we have limited our prophylactic treatment to a single dose of 2 cc of vaccine and have infected two weeks later with 1 cc of a 1:50 dilution of street virus administered intramuscularly in the loin.

Potency Tests

Table I indicates the results obtained in our initial test with a group of rabbits which had received two injections of vaccine prepared from a strain of fixed virus in use in the Philippine Islands. This vaccine was injected 48 hours subsequent to its preparation. The street virus used for the infection was a strain recovered from a natural case of rabies in a dog in Manila. It had been passed successively through two rabbits before use in the experiment.

Table I—*Results of initial test*

Rabbit	Amount of Vaccine (cc)	Street Virus (1:100) Administered Subdurally Two Weeks Subsequent to Vaccination (cc)	Results
1	2.0	0.1	Survived
2	2.0	0.1	Survived
3	2.0	0.1	Died immediately after inoculation of virus
4	2.0	0.1	Survived
5	2.0	0.1	Died of rabies 22 days after infection
6	2.0	0.1	Survived
7	2.0	0.1	Died of rabies 20 days after infection
8	2.0	0.1	Survived
9	None	0.1	Died of rabies 19 days after infection
10	None	0.1	Died of rabies 20 days after infection

In this test one of the 8 vaccinated animals died, as an immediate result of the subdural injection of virus. Of the 7 remaining, 5 resisted the infection and 2 died of rabies. Two rabbits employed as controls both died of rabies.

Table II shows the results obtained with a lot of vaccine prepared in this country.

TABLE II—*Results with vaccine prepared in Washington*

RABBIT	AMOUNT OF VACCINE (CC)	STREET VIRUS (1:100) ADMINISTERED SUBDURALLY TWO WEEKS SUBSEQUENT TO VACCINATION (CC)	RESULTS
1	2.0	0.1	Survived
2	2.0	0.1	Died from broken back
3	2.0	0.1	Survived
4	2.0	0.1	Survived
5	2.0	0.1	Died of rabies 23 days after infection
6	2.0	0.1	Died of rabies 18 days after infection
7	2.0	0.1	Survived
8	2.0	0.1	Died of rabies 17 days after infection
9	2.0	0.1	Survived
10	2.0	0.1	Died within 1 hour after inoculation of virus
11	2.0	0.1	Died of rabies 15 days after infection
12	2.0	0.1	Survived
13	2.0	0.1	Died of rabies 20 days after infection
14	2.0	0.1	Survived
15	2.0	0.1	Died of rabies 19 days after infection
16	None	0.1	Died of rabies 13 days after infection
17	None	0.1	Died of rabies 17 days after infection
18	None	0.1	Died of rabies 15 days after infection

In this series we lost one of the 15 vaccinated rabbits as a result of a broken back sustained in handling and a second as an immediate result of the subdural injection of virus. Of the 13 remaining vaccinated animals, 7 were sufficiently protected to survive the subdural infection. Six developed rabies and died as a result thereof. We employed 3 controls, all of which developed the disease.

I am not going to take time to present tabulations of several additional experiments in which we administered two doses of vaccine to each rabbit in the several series and later infected them subdurally. Suffice it to say that our results in the main were similar to those depicted in the preceding table. We protected against subdural infection approximately half of the rabbits we inoculated.

Realizing that a single injection of vaccine, if it afforded ample protection against natural infection, would greatly facilitate canine vaccination on a large scale, we carried out a number of tests in which the rabbits were given but one injection of vaccine. One of these series of rabbits was subsequently infected subdurally, whereas in the others the intramuscular method of infection was followed.

Table III indicates the results with 15 vaccinated animals and 3 controls.

TABLE III—*Results of tests on 15 rabbits with 3 controls*

RABBIT	AMOUNT OF VACCINE (CC)	STREET VIRUS (1:100) ADMINISTERED SUBDURALLY TWO WEEKS SUBSEQUENT TO VACCINATION (CC)	RESULTS
1	2.0	0.1	Died of rabies 16 days after infection
2	2.0	0.1	Died of rabies 22 days after infection
3	2.0	0.1	Survived
4	2.0	0.1	Survived
5	2.0	0.1	Died of rabies 21 days after infection
6	2.0	0.1	Survived
7	2.0	0.1	Died within 24 hours after subdural infection
8	2.0	0.1	Died of rabies 19 days after infection
9	2.0	0.1	Died of rabies 20 days after infection
10	2.0	0.1	Survived
11	2.0	0.1	Died of rabies 19 days after infection
12	2.0	0.1	Died of rabies 19 days after infection
13	2.0	0.1	Died of rabies 20 days after infection
14	2.0	0.1	Survived
15	2.0	0.1	Survived
16	None	0.1	Died of rabies 19 days after infection
17	None	0.1	Died of rabies 17 days after infection
18	None	0.1	Died of rabies 20 days after infection

In this series of rabbits the one 2-cc dose of vaccine protected 6 out of 14 vaccinated animals, the fifteenth animal dying as an immediate result of the subdural inoculation. The three controls all died of rabies.

Table IV indicates the results obtained with a series of 17 rabbits. Twelve of these animals each received a single dose of 2 cc of vaccine and subsequently an intramuscular infection of street virus. The five other rabbits in this group were employed as controls. Inasmuch as intramuscular infection, in rabies, is not so certain as the subdural route, a larger number of controls were employed.

In this test 10 of the 12 vaccinated rabbits survived. Of the controls, 3 developed rabies and 2 failed to develop the disease.

A repetition of this test, depicted in table V, gave even more striking results, in that all of the 12 vaccinated animals survived, whereas 4 out of the 5 controls developed rabies.

We have repeated the tests depicted in tables IV and V a number of times and have uniformly protected a large percentage of the vaccinated animals. We have not, however, with the

TABLE IV—*Results of tests on 12 rabbits with 5 controls*

RABBIT	DOSE OF VACCINE (CC)	STREET VIRUS (1:50) ADMINISTERED INTRAMUSCULARLY TWO WEEKS SUBSEQUENT TO VACCINATION (CC)	RESULTS
1	2.0	1.0	Survived
2	2.0	1.0	Survived
3	2.0	1.0	Survived
4	2.0	1.0	Survived
5	2.0	1.0	Survived
6	2.0	1.0	Died of rabies 25 days after infection
7	2.0	1.0	Survived
8	2.0	1.0	Survived
9	2.0	1.0	Survived
10	2.0	1.0	Died of rabies 21 days after infection
11	2.0	1.0	Survived
12	2.0	1.0	Survived
13	None	1.0	Died of rabies 20 days after infection
14	None	1.0	Survived
15	None	1.0	Survived
16	None	1.0	Died of rabies 21 days after infection
17	None	1.0	Died of rabies 23 days after infection

TABLE V—*Repetition of test (table IV)*

RABBIT	DOSE OF VACCINE (CC)	STREET VIRUS (1:50) ADMINISTERED INTRAMUSCULARLY TWO WEEKS SUBSEQUENT TO VACCINATION (CC)	RESULTS
1	2.0	1.0	Survived
2	2.0	1.0	Survived
3	2.0	1.0	Survived
4	2.0	1.0	Survived
5	2.0	1.0	Survived
6	2.0	1.0	Survived
7	2.0	1.0	Survived
8	2.0	1.0	Survived
9	2.0	1.0	Survived
10	2.0	1.0	Survived
11	2.0	1.0	Survived
12	2.0	1.0	Survived
13	None	1.0	Died of rabies 24 days after infection
14	None	1.0	Survived
15	None	1.0	Died of rabies 22 days after infection
16	None	1.0	Died of rabies 19 days after infection
17	None	1.0	Died of rabies 23 days after infection

intramuscular mode of infection, been able to produce rabies in all control animals. The incidence among the control animals has varied between 40 and 80 per cent. There has, however, been such a very marked and wide difference between the incidence of the disease in vaccinated and control animals as to leave no doubt as to the protection afforded by the vaccine.

The Active Principle of the Vaccine

In experiments with rinderpest the writer demonstrated that when rinderpest virus, contained in the solid tissues of animals slaughtered in the acute stage of rinderpest, is freed from such tissues and killed by chloroform (or other means), it is of no value as an immunizing agent even though large quantities of such killed virus be administered. On the other hand, fine emulsions of these tissues, in which the virus has been killed by the use of chloroform, are highly efficacious for the immunization of susceptible animals against rinderpest. In our work with rabies vaccine we have found an identical situation. Rabies vaccine containing killed virus, when freed of its tissue content, immediately loses its value as an immunizing agent. Thus, in rinderpest and rabies we note that the killed viruses of these diseases, when free of tissue particles, are of no demonstrable value for immunization purposes.

Tables VI and VII indicate the results obtained with two fractions of a chloroform-treated rabies vaccine. This lot of vaccine was subjected to 5000 atmospheres of pressure and the fluid thus

Table VI—*Vaccination results with chloroform-treated fixed virus filtrate "R-4"*

Rabbit	Amount of Vaccine (cc)	Street Virus (1:100) Administered Subdurally Two Weeks Subsequent to Vaccination (cc)	Results
VF-1	2.0	0.1	Died of rabies 13 da. subsequent to infection
VF-2	2.0	0.1	Died of rabies 22 da. subsequent to infection
VF-3	2.0	0.1	Died of rabies 19 da. subsequent to infection
VF-4	2.0	0.1	Died of rabies 17 da. subsequent to infection
VF-5	2.0	0.1	Died of rabies 16 da. subsequent to infection
VF-6	2.0	0.1	Died of coccidiosis 5 da. after rabies infection
VF-7	2.0	0.1	Died of rabies 15 da. subsequent to infection
VF-8	2.0	0.1	Died of rabies 17 da. subsequent to infection
CF-1	None	0.1	Found dead morning following subdural injection
CF-2	None	0.1	Died of rabies 16 da. after infection

obtained was then passed through a sterile paper filter and was designated "Filtrate R-4." The pressed residue was then triturated in sufficient sterile physiological saline solution to bring it back to the original volume of the untreated vaccine.

TABLE VII—*Vaccination results with chloroform-treated fixed virus residue "R-4"*

RABBIT	AMOUNT OF VACCINE (CC)	STREET VIRUS (1:100) ADMINISTERED SUBDURALLY TWO WEEKS SUBSEQUENT TO VACCINATION (CC)	RESULTS
V-1	2.0	0.1	Survived
V-2	2.0	0.1	Survived
V-3	2.0	0.1	Died of rabies 19 da. subsequent to infection
V-4	2.0	0.1	Survived
V-5	2.0	0.1	Died within 2 hrs. following subdural inoculation
V-6	2.0	0.1	Survived
V-7	2.0	0.1	Died of rabies 16 da. subsequent to infection
V-8	2.0	0.1	Survived
C-1	None	0.1	Died of rabies 14 da. subsequent to infection
C-2	None	0.1	Died of rabies 18 da. subsequent to infection

It will be noted that none of the rabbits injected with the vaccine filtrate were protected against artificial infection. On the other hand, the residue protected 5 out of 7 rabbits.

As pointed out in the report on the rinderpest work, it is my opinion that the active principle of the rinderpest vaccine represents something in which tissue activity is concerned. This "something" may be a cellular principle resulting from the reaction between the virus and tissue cells or it may be a particular "stage" of the virus which occurs in the tissue cells. Whatever it is, it is firmly linked with the tissue cells and is not capable of being readily separated from them by ordinary methods. Our evidence indicates that a similar situation exists in rabies.

CONCLUSIONS

1. A chloroform-treated rabies vaccine containing 33⅓ per cent brain tissue has been found to be a very efficacious immunizing agent against the disease.

2. A single dose of 5 cc for dogs should prove effective against natural infection.

3. It is believed that the active principle of this vaccine is something other than dead virus. It is possibly a cellular prin-

ciple resulting from the reaction between the virus and tissue cells or it may be a particular "stage" of the virus which occurs only in the tissue cells. Whatever it is, it is firmly linked with the tissue cells and is not capable of being readily separated from them by ordinary methods.

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"Out of the Shadows" Enters Film Valhalla

After nearly ten years of effective service in the fight against bovine tuberculosis, "Out of the Shadows," one of the early educational films made by the U. S. Department of Agriculture, has been laid on the shelf. The last remaining copy of more than forty, once in circulation, has been withdrawn—battered, "rainy," and full of torn sprocket-holes—and no more copies will be printed. "Out of the Shadows" has served its day, and its negative now goes into the archives of the Department, with other old films honorably discharged from service.

No other film circulated by the Department has made a record approaching that of "Out of the Shadows." It has been used in every anti-tuberculosis campaign waged since 1921, and has been shown in almost every rural community in the United States where dairy cattle are important. Released in 1921, 17 copies were put into circulation that year; in 1922, 12 more copies were put into circulation; and in 1923, as many more. In all, 42 copies have been released. These have been shipped into the field, 1,644 times, bookings for a single copy frequently running several weeks.

Sixty-one copies of this film have been sold for use in foreign countries, including Uruguay, Belgium, Guatemala, the Union of South Africa, and Great Britain.

"Out of the Shadows" was directed by F. W. Perkins, formerly chief of the Office of Motion Pictures. Photography was by G. R. Goergens, who is still with the Department. The story was by Drs. J. E. Gibson and L. B. Ernest, of the Tuberculosis Eradication Division, both of whom are known personally by thousands of veterinarians throughout the country.

CLOSTRIDIUM WELCHII INFECTION IN THE DOMESTICATED FOWL*

By K. W. NIEMANN, *University of Nevada, Reno, Nev.*

Six single-comb White Leghorns, apparently twelve to sixteen weeks of age, were received at our laboratory, August 19, 1929. They were accompanied by a letter from the owner to the effect that he had lost more than 100 of them, seemingly from what he termed a combination of ailments. We learned also that there were 500 to 600 in the flock, all of the same age and from the same lot of chicks. They had been previously put on a light tobacco-dust mash, and at this time had been put on a mash containing 40 per cent dried milk and 4 per cent tobacco dust.

Upon visiting the ranch some time later, we observed that sanitary conditions were not of the best, although the owner informed us to the effect that he had been raising birds there for seventeen years with no previous trouble.

Clinical symptoms were those which might be expected in cases of parasitism or partial inanition—ruffled feathers, bleached beak and shanks, and slight emaciation.

Autopsy findings were noted as follows:

Bird 1: Autopsied, 8-19-29. Beak and shanks appeared bleached. Respiratory tract was normal. Circulatory system normal. No nutritional nodules in esophagus. Marked catarrhal enteritis.

Bird 2: Autopsied, 8-20-29. Beak and shanks appeared bleached, definite emaciation present. Respiratory tract normal. Circulatory system normal. No nutritional nodules observed in esophagus. Mild inflammation of intestinal mucosa; gelatinous material in lumen. Kidney tubules and ureters filled but not distended with urates.

Bird 3: Autopsied, 8-20-29. Beak and shanks definitely bleached. Respiratory tract normal. Circulatory system normal. No nutritional nodules in esophagus. Catarrhal enteritis; one cecum worm observed; gall-bladder markedly distended.

Bird 4: Autopsied, 8-27-29. Respiratory tract normal. Circulatory system normal. Numerous caseous necrotic appearing nodules were observed throughout the intestinal tract, which were macroscopically almost indistinguishable from those found in cases of acute miliary avian tuberculosis. A marked enteritis was present. The kidneys were greatly swollen and contained caseous necrotic areas. The spleen appeared normal. Liver smears were negative for acid-fast rods but a short, thick rod was observed.

Birds 5 and 6 were held for observation during which time bird 6 developed symptoms of posterior paralysis.

Bird 5: Autopsied, 9-20-29. Respiratory tract normal. Circulatory system normal. Marked enteritis. *A. lineata* infestation and marked

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H. papillosa infestation. Coccidia demonstrated by glycerin-flotation method. Left sciatic nerve greatly enlarged and right sciatic nerve somewhat enlarged.

Bird 6. Autopsied, 9-20-29. Bird emaciated and paralyzed. Respiratory tract normal. Circulatory system normal. Marked enteritis and taeniasis. *H. papillosa* present. Coccidia demonstrated by glycerin-flotation method.

Aerobic bacteriological examinations from heart-blood and liver of all specimens were negative. Anaerobic bacteriological examinations of the liver of bird 4 and the heart-blood of bird 6 yielded cultures which gave reactions in fourteen differential carbohydrate media identical to *Clostridium welchii*, Type II, as described by Bergey.

The method of isolation was as follows: Original inoculations were made by means of sterile capillary pipettes into freshly boiled beef heart-peptic digest media. Peptic digest-agar shake dilutions were then made after incubation for 24 hours. Deep-agar colonies were picked from the sliced shake dilutions with sterile capillary pipettes, after incubation for 24 hours, and transferred to freshly boiled beef heart-peptic digest. This was repeated until four consecutive sets of agar-shake dilutions had been made and colonies picked.

Upon inoculation, cultures obtained from both birds proved virulent to cavia. The cavia inoculated with the culture from bird 4 developed a marked miasm of the lesion before death. It showed a necrosis of the connective tissue extending from the point of inoculation in the hind leg to the submaxillary region. Small anemic infarcts were present on the liver. Both aerobic and anaerobic secondary invaders had developed to such an extent that isolation of the original organism was not feasible. This same culture also proved virulent for a chicken, 4.0 cc of a 24-hour beef heart-peptic digest culture producing death in 24 hours. This bird showed coagulation necrosis of the breast muscles and the sartorius muscles, being most marked on the side of inoculation. Necrosis of a considerable portion of the parietal surface of the left lobe of the liver was present, together with necrotic nodules throughout the liver similar to those occurring in the original specimen. The liver also showed a number of anemic infarcts. The kidney showed evidence of beginning infarct areas. The peritoneum on the left side of the gizzard was necrotic. Fibrinous pericarditis was present.

Immediately after isolation, the culture from bird 6 proved avirulent for chickens. Eleven months later, after passage

through a series of four deep-agar shakes for purification and two transfers through beef heart-peptic digest media containing iron, 5 cc of a nine-hour culture intraperitoneally killed a healthy bird in less than twelve hours. Due to the unsuspected virulence and consequent excessive dose, the bird did not present the expected postmortem picture. Examination showed arborization and injection of the blood-vessels of the breast musculature, giving a mottled appearance to the breast. There was marked edema of the intramuscular tissues especially medial to the thighs. A bloody transudate in the abdominal and thoracic cavities was a rather prominent feature. An extensive fibrinous peritonitis caused marked adhesions. A necrotic appearing area, about 7.5 mm. in diameter, was located antero-medially on the right lobe of the liver. There was no evidence of an intestinal puncture. Fibrinous pericarditis was not marked. Lung edema was very marked.

Culture feeding failed to infect healthy birds shortly after the organism was isolated. After eleven months, 2 cc of a ten-hour culture intravenously proved non-infectious to healthy birds.

Histologically the early lesions of the field case presented a picture of necrobiosis in its stricter sense. Both kidneys and liver exhibited passive congestion to a marked extent. There appeared considerable exfoliation and degeneration of renal epithelium. Vacuolization, although present in the kidneys, appeared most marked in the liver lesions. It would appear that the organism in the fowl tends to produce focal areas of necrosis and is reluctant to become bacteriemic.

Summary and Conclusions

1. *Clostridium welchii* was isolated from two of six birds from a badly parasitized flock.

2. The organism was isolated, identified, caused to reproduce the condition by inoculation, and recovered from the isolated bird in establishing the etiology of the infection.

3. The infection atrium in this case appeared to be the intestinal tract.

4. It would appear from this study that *Clostridium welchii* infection in the domesticated fowl is not a specific disease entity but a sporadic infection that may occur in a heavily parasitized flock.

THE UNIVERSITY OF CALIFORNIA COMMITTEE FOR THE STUDY OF BRUCELLA INFECTIONS*

By C. M. HARING, *Berkeley, Calif.*

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In November, 1929, at an informal conference of some members of the medical and agricultural faculties of the University of California, it was decided to call a meeting of those persons in the University who were working on undulant fever in man and related diseases in animals. This meeting was held, and attended by representatives of the Departments of Bacteriology, Berkeley; the Hooper Foundation for Medical Research, in San Francisco; the Division of Veterinary Science, in Berkeley, and the Divisions of Animal Husbandry and Veterinary Science, in Davis.

In accordance with decisions made at that meeting, arrangements were made with the President of the University to appoint a committee for the study of Brucella infections, with Dr. K. F. Meyer as chairman and Dr. C. M. Haring as secretary. The Committee included not only members from the University departments interested, but representatives of official and professional activities throughout the State which were concerned with the Brucella problem. Representatives of the Health Departments of Arizona and Oregon and of the Oregon Agricultural Experiment Station also were invited to serve on the committee. As a result, the work on brucelliasis in the University has been correlated with the work of other agencies at some five points: the control measures for Bang's disease, of the Division of Animal Industry, State Department of Agriculture; the epidemiological work on undulant fever, of the State Department of Public Health; the study of cases of undulant fever, in certain hospitals and clinics in the State; and with the work of certain medical and veterinary practitioners, particularly those doing inspection work for the County Medical Society Milk Commission.

The first tangible result of the work of the Committee was a pamphlet entitled, "Undulant Fever or Brucelliasis," published as Special Bulletin No. 50, by the California Department of Public Health. This bulletin treats of the history, epidemiology,

*Presented at the sixty-seventh annual meeting of the American Veterinary Medical Association, Los Angeles, Calif., August 26-29, 1930.

clinical characteristics and control measures for undulant fever and may of obtained free by writing to the State Department of Public Health at Sacramento.

Recognizing the necessity of sufficient uniformity in technic to make comparable results in agglutination testing in various laboratories in the State, a sub-committee worked long and hard to secure an agreement in the procedures used by the various technicians concerned. The result is a mimeographed guide, entitled, "Diagnostic Laboratory Procedures," which is being sent to laboratories that are doing agglutination testing in California. Copies of this may be secured free by writing to the Division of Veterinary Science, University of California, Berkeley.

During the past six months, important work has been started by certain members of the Committee. One project is to determine the variability of the main types of the Brucella group in various species of animals. Participants in this project include Drs. K. F. Meyer and B. U. Eddie, of the Hooper Foundation; F. M. Hayes and J. A. Howarth, of the University Farm, at Davis; and J. Traum, B. S. Henry and C. M. Haring, of the Division of Veterinary Science, Berkeley. Among the cultures to be studied, it is hoped that freshly isolated strains may be obtained as follows:

Brucella melitensis of human origin
Brucella melitensis of caprine origin
Brucella suis of human origin
Brucella suis of porcine origin
Brucella abortus of human origin
Brucella abortus of bovine origin

It was recognized that cultures of the Brucella group more often cause accidental laboratory infections than most pathogenic bacteria, not only in persons but also in experiment animals in the laboratory. Therefore, arrangements were made to prevent confusion, by carrying on the work, as far as possible, with melitensis at the Hooper Foundation for Medical Research in San Francisco, with porcine strains at the University Farm at Davis, and with bovine strains at Berkeley.

Variability studies on a culture isolated by Hayes from swine at the University Farm is the only part of this project yet under way. Cattle, swine, sheep, goats and fowls have been inoculated, and in all cases evidence of infection has been obtained. Two

cows and one goat became carriers, and milk from each of these animals was used to feed monkeys. The only monkey to show infection received milk from one of the goats. The porcine strain (55), so called, which was used in the experiments at Davis, had been isolated about six months previous to the use of its transplants for inoculation of the various animals. At the time of inoculation, the cultures showed about 85 per cent "S" type colonies, and 15 per cent "R" type. Since the inoculations, several cultures have been obtained from the cattle, goats, swine and fowls, and in all cases have shown 100 per cent "S" type colonies. This work is not yet completed.

Another project under way is an epidemiologic study of undulant fever, by Dr. J. C. Geiger, Professor of Epidemiology, University of California, in cooperation with the California Department of Public Health. Observations which they are making have been correlated with work on Bang's disease conducted by Dr. K. G. McKay, Agricultural Extension Specialist, University of California, Berkeley; Dr. J. A. Howarth, Associate in Veterinary Science, University Farm, Davis, and other veterinarians in the University. At this present meeting of the American Veterinary Medical Association, two papers* on brucelliasis will give results of some of this work.

At the time the Committee was organized, it happened that the State Division of Animal Industry had begun a project at the request of the Del Norte County Board of Supervisors, to compel the segregation of all dairy calves from tuberculous cows in that county within 48 hours of their birth. The work was being carried out under section 12 of the California bovine tuberculosis law. A majority of the dairymen of the County had requested that the University cooperate in efforts to eradicate Bang's disease as well as tuberculosis, and Dr. K. G. McKay was assigned to this by the University. He has taken blood samples from practically all cows in the county; and of about 5000 tests, approximately 17 per cent reacted. Through the cooperation of physicians in Del Norte County, Doctors Geiger and Meyer have arranged to secure blood samples from as many persons in the County as possible.

At the Hooper Foundation, arrangements also have been made to test herdsmen, milkers, veterinarians, meat inspectors

*Howarth, J. A., and Hayes, F. M.: Brucelliasis in Swine at the University of California, with an Epidemiological Report of the Infection of Two Attendants. Geiger, J. C.: Epidemiologic Data of Brucelliasis—Report of Two Human Cases. (To be published in the JOURNAL.)

and persons engaged in the handling of raw meat and milk supplied to the city of San Francisco. The data are gradually accumulating, and a correlation is to be made between the epidemiology of undulant fever and the epizoology of Bang's disease, which may yield interesting results.

Another project is the cooperative study of the thermal death time of Brucella organisms, which was started July 1, 1930, between the Hooper Foundation for Medical Research and the Division of Veterinary Science. A study of the literature showed the publications available on thermal death time of Brucella to be meagre and contradictory. Arrangements were made to have the work done at the Research Laboratory of the National Canners Association, in San Francisco, as this laboratory was operating under the direction of the Chairman of the University Brucella Committee, and had the best facilities for thermal-death-time work of any California laboratory.

Brucella infections are known to have been enzootic in goats in certain sections of the southwestern United States for several years. However, little information has been available regarding the types of organism predominating there. A survey has been begun in Arizona by members of the staff of the Hooper Foundation. Several hundred serum specimens have been examined. A shedder goat has been brought from Arizona to San Francisco and the organisms isolated from it are being studied.

At the veterinary science laboratory in Berkeley, studies on immunity and the carrier problem in dairy cattle have been under way for several years. Publications are now available giving the results of past work, and additional data will be published by the California Agricultural Experiment Station in the near future.

Since January, 1929, observations have been made at a large dairy where Bang's disease has been slowly spreading, because it has been impracticable to isolate completely or remove all the infected cattle. Extensive bacteriologic and serologic records obtained in this herd are being correlated with the breeding, gestation and lactation records of the cows and the amounts of milk and butter fat produced.

Already the beneficial influence of the University of California Committee for the Study of Brucella Infections has improved the relations of the various agencies throughout the State which are concerned with the undulant fever and Bang's disease problems. Progress has been made in unifying and

improving diagnostic technic. The research work of certain University departments has been correlated and the individual members of the Committee have been privileged to attend monthly meetings and exchange ideas, discuss their work with one another, and make plans for future cooperative projects. The committee meetings usually begin about 5 o'clock and continue until late in the evening. The discussion proceeds throughout the dinner and resembles that of a university seminar, in that the latest literature on brucelliasis is discussed. The voluminous correspondence by the Chairman with foreign investigators has been made available to all at each meeting and the mimeographed minutes of the meetings have been amplified to include important information regarding brucelliasis throughout the world.

This description of committee work has been presented in the hope that groups of investigators in other states may find some helpful suggestions in it for cooperative organization to combat diseases of man or animals.

St. Johnswort Seldom Kills Stock

St. Johnswort, long considered a plant poisonous to cattle and sheep, seldom causes death and is not likely to be a source of much loss to stockmen because of its toxic properties. This information is the result of a series of feeding experiments carried on near Salina, Utah, by C. D. Marsh and A. B. Clawson, of the U. S. Department of Agriculture. Details of the investigations are given in Technical Bulletin 202-T, "Toxic Effects of St. Johnswort (*Hypericum perforatum*) on Cattle and Sheep," just issued by the Department.

The cattle and sheep used in the investigations, which covered four years, were slightly affected by eating the plant, when it was mixed with hay. However, the poisonous effects were not often pronounced and then only after large feedings.

Cases of sickness and even death of domestic animals had been attributed to this plant, but experimental evidence regarding its toxic character was lacking until the investigations were conducted. In California and Oregon the plant is known as Klamath weed, and in the former state its abundance has been of concern to many stock-owners who depended on the ranges for late fall feed. St. Johnswort occurs to a limited extent in eastern states.

Technical Bulletin 202-T may be procured from the Office of Information, U. S. Department of Agriculture, Washington, D. C.

SOME OF THE USES OF NORMAL SALINE SOLUTION INFUSION IN CANINE PRACTICE*

By H. Calvin Rea, *Charlotte, N. C.*

In 1861, the famous English surgeon, Lord Lister, published an article advocating antiseptic surgery. A short time later, he advised that wounds be covered with gauze, impregnated with carbolic acid. Very soon thereafter, surgeons began bathing skin wounds with salt solution; great pans of salt water were poured into the bellies which contained pus following laparotomies. Since then it has been used continuously; constantly growing in its usage until today it is admittedly one of the most valuable agents known to medical science. Several substitutes for, or additions to, salt solution have reached some practical importance, such as glucose, sodium bicarbonate, acacia and gelatin.

The terms "normal" saline solution and "physiological" saline solution are used synonymously by most authors. However, properly, a normal solution is one which contains in one liter sufficient of the dissolved substance to replace one gram of hydrogen. A normal solution of sodium chlorid contains, therefore, 58.37 grams per liter, or 5.837 per cent, which is approximately ten times the strength of a physiological salt solution, usually called, incorrectly, a normal salt solution. In about one hundred cases we have used Locke's balanced solution, which is chemically pure chlorid of sodium, potassium and calcium, but the results do not indicate a greater clinical value than a mere sodium chlorid. A .9 per cent sterile solution of chemically pure sodium chlorid, which is isotonic to mammalian blood, is commonly called normal saline solution and will be considered as such herein.

How Action is Brought About

Even though much experimenting has been done, it has never been definitely determined how salts bring about their action in the body. Wilcox says:

> Sodium chlorid has practically no specific action. Its effects are limited to the alteration in the fluids produced by its excess or deficiency and they present a typical example of what is known as "salt action." As its molecular weight is small and as it dissociates rapidly into two ions it possesses great osmotic power.

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Quoting from Solman:

> If a foreign solution is introduced, f. i., into intestines or peritoneal cavity or blood—the osmotic forces very rapidly bring its total molecular concentration to that which is normal for the tissues. . . . In general the exchange occurs mainly in the direction which will favor the normal physiologic functions, i. e., in direction of absorption. . . . In whatever concentration, and by whatever channel salts are introduced into the body, they tend to increase the quantity of blood, or rather the plasma, and to render it more watery and less viscid; in other words to produce hydremic plethora.

Smith states:

> The salts in the body perform important functions in connection with secretions and excretions. As Foster expresses it, they direct the metabolism of the body, though how they do so is unknown. To their presence is due the normal composition of the body fluids and tissues, for they regulate the water flow from blood to tissues and vice versa. When a deficiency in salts occurs, the body apparently for some time draws on its own store and then nutritive changes follow.

The sodium salts in the blood and lymph take the principal part in maintenance of normal osmotic pressure. Salts exert a regulating influence upon the rate of heart beat. They contribute no energy to the body, but their function in nutrition is of utmost moment.

Salt Action on the Urine

Where large amounts of this solution are administered, the kidneys are flushed within about thirty minutes; saline diuresis would therefore result in a "flushing of the body." Creatinine is eliminated first; urea nitrogen, second; uric acid, third; non-protein nitrogen, fourth (Reinhart).

Indications

In most instances saline infusions are used to tide the patient over an acute condition, such as the following:

Severe hemorrhage.
Dehydration.
Shock—traumatic or surgical.
Acute toxemia.
Acute intestinal obstruction.
Acute gastritis.
Acute gastroenteritis.
Pneumonia.
Septicemia.
Nephritis (in the absence of edema).
Various metabolic disturbances.
Feeble heart action and low vascular tension.
Depletion of blood chlorids.
Acute vomition and vomiturition.

Contraindications

Edema.
Leaky heart valve.
Intraperitoneally, in peritonitis.

Apparatus and Technic

A 1000-cc graduate glass irrigator equipped with several glass Y's and needles and a stand for same, in addition to other hospital equipment.

Technic must be aseptic. In all administrations the solution should enter the body very slowly, particularly in intravenous and subcutaneous routes. The irrigating stand should be raised only slightly above the level of the patient. The solution should enter the body at a temperature of 103° to 105° F.

Modes of Introduction

1. Intraperitoneally: This is the method of choice, provided there is no pertionitis. Only one point of entrance is used. It is more simple and can be given more rapidly and with least amount of discomfort to the patient.

2. Subcutaneously: In this method several points of entrance should be used; this being made possible by use of the glass Y's. The only objection to this method is the time element, which is of great importance to the busy practitioner. Subcutaneous infusions should cease when manifestations of pain or marked discomfort are in evidence. The amount given an unconscious patient is determined by experience.

3. Intravenously. There are two reasons why the writer does not recommend this method. First, because of the danger of distension of the heart; second, because in the majority of cases in which normal saline is indicated the veins are in a marked degree of collapse, making their invasion difficult. The saphenous vein is the one of choice.

Dosage

The dosage depends on the following:
Size of animal.
Condition of patient.
Rate of absorption.
Comfort of patient.
Mode of introduction.

Frequent infusions are for various reasons more effective than large doses. The tissues have a peculiar affinity for this sub-

stance. Therefore, the time to repeat the dose is self-evident. Two to three liters within a twenty-four-hour period, given at four- to eight-hour intervals, for a forty-pound dog, is a fair working basis. Due to the fact that intake of salt increases the osmotic pressure of the tissues, care should be taken not to produce an edema. The tissues should be respected under all circumstances.

Sequelae

Infection following faulty technic.
Edema.
Overdistension of the heart.
Glycosuria.
Rise of temperature.

Table I—*Case reports*

Diagnosis	Cases	Recoveries	Mortality Rate (%)
Acute toxemia	93	80	14.0
Black tongue	86	74	14.0
Operative procedures	76	66	13.1
Accident cases	58	44	24.1
Distemper	44	32	27.1
Acute intestinal obstruction	18	14	22.1
Acute gastritis Acute gastroenteritis	68	60	12.0
Pneumonia	32	25	22.0
Severe hemorrhage	28	24	14.1
Miscellaneous	48	42	12.0
Totals	551	461	Aver. 16.3

Remarks

Until about eight months ago, our dosage of normal saline solution for a forty-pound dog did not exceed 600 cc during a twenty-four-hour period and even that was administered in most cases as a last-resort measure. The results indicated a definite clinical value in only a limited number of conditions and a small percentage of cases. A change in time of administration and dosage proved so gratifying that it became a regular hospital routine. It has also proven to be of value in a greater variety of conditions. Treatment begins immediately when indications appear or upon entrance to hospital and continued daily, not infrequently six to ten days. Since time does not permit a detailed comparison of the mortality rate in the above case reports, to the rate prior to this, it would be impossible to make a positive statement, because many conditions influence the

outcome of our cases. Admittedly it is high, but the report includes all cases within an eight-month period, both favorable and unfavorable.

In February, 1929, the writer reported 112 cases of black tongue with a mortality rate of 30 per cent. No material change was made in the treatment of 86 cases above reported, except that the use of saline was added.

Conclusions

1. Normal saline solution in canine practice is an invaluable therapeutic agent. Many cases which enter the small-animal hospital for treatment are critically ill, and this solution, better than anything we know of, tends to tide the patient over various crises until the cause of the condition can be removed.
2. The dosage, covering a period of twenty-four hours, must of necessity be larger than that previously given by us.
3. Instead of waiting to use saline as a last-resort measure, it should be given as early as possible.
4. Treatment should be continued until the patient seems out of danger, continuing oftimes as long as ten days.
5. We have never experienced one of the sequelae mentioned above, except a slight rise in temperature.
6. Our usage of normal saline has materially broadened in recent months, covering a greater number of conditions.
7. Compared with other remedial agents, normal saline has few contraindications.
8. The dangers incident to saline administration are few and do not exist within reasonable bounds.
9. In most conditions in which saline is indicated it has no peer and no generally successful substitute.
10. Its value is unquestioned by our most learned men in all branches of medicine.

Appointed

The following Manitoba veterinarians recently passed the Civil Service examination for veterinary inspectors in the Dominion Department of Agriculture and received appointments in the Meat Inspection Division, at Winnipeg: Drs. A. J. Andries, Deloraine; Laurence Bailey, Rivers; W. C. Hodgins, Oak Lake; J. S. Bowie, McGregor; J. C. McConnell, Roblin, and A. S. Frame, Miami.

BULLA-OSTEOTOMY IN THE DOG*

By G. W. McNutt *and* J. E. McCoy

The State College of Washington

Pullman, Washington

The need for bulla-osteotomy was first called to the attention of one of the writers following the first severe outbreaks of swine "flu" in the Middle West (1917-1921) and the complication of otitis media which occasionally followed. Otitis media was seen also in the calf, the cat, and the dog. Involvement of the cells of the bulla ossa was not an infrequent occurrence. These cases were acute when compared to the conditions more recently seen in dogs in the Washington State clinic.

Any operation upon the bulla ossea was at first thought practically impossible because of the deep position of the bulla and the network of arteries and nerves which would have to be penetrated, and yet not injured.

The conditions in dogs presented at our clinic (Washington) during the last few years have made it imperative that something be done in this direction.

The operation is not the equivalent of the mastoid operation in the human, but is far more difficult, because the osseous spaces involved are ventral and deep in animals, instead of posterior and superficial as in man.

Briefly stated, the indication for bulla-osteotomy is suppurative inflammation of the middle ear.

The frequency of the occurrence of this condition varies greatly in different parts of the country, as the causes exist in varied degrees in different localities. In our own territory we find that the head and awns of foxtail and cheet grass are the most frequent causes, while in other localities, factors favoring the development of otitis media may be climatic conditions, together with any irritant, such as neglected infestations with ear mites or otitis externa. Any progressive inflammatory process extending from the nasal passages and throat up the Eustachian tubes also may be a factor.

Dr. E. A. Ehmer, of Seattle, Washington, reports a very interesting type of otitis which he says may be either otitis

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externa or otitis media. It is caused by, or at least associated with, "ear polypus." The growth is vascular in character and has but little definite histological structure. It arises from a small base, usually in the external ear, but sometimes in the middle ear, and has a tendency to grow very rapidly and to fill the entire canal. Complete surgical removal with thorough cauterization is the only known successful treatment. It is impossible to get at the base, where these conditions are deep-seated or in the middle ear. The base of some of these may be exposed by bulla-osteotomy.

SYMPTOMS

The symptoms of otitis media in its acute and subacute forms are: frequent scratching the ears and shaking the head from side to side, and carrying the head to one side a part or all of the time. Incoordination is shown most markedly when the patient is first asked to move after lying down, or being asleep. Frequently, this symptom soon passes off and is observed only when the dog makes an effort to turn suddenly or tries to react quickly to some sudden or sharp command.

Closer examination reveals some tenderness on palpation over the bulla ossea, some enlargement of the lymph-glands in the pharyngeal region, and occasionally an irritating, hacking cough. The discharge from the ear is purulent, rather sanguineous, and has a necrotic odor. At times it is quite profuse and at other times rather slight. The auditory canal always shows a catarrhal inflammatory change, with a gray membranous discharge on the surface, under which the injected, slightly swollen membrane may be exposed by the use of a cotton swab. If the tympanic membrane is present, or a portion of it still remains, it will show definite injected blood-vessels, and occasionally there will be crusts accumulated on its edge or surface. Still deeper, in the middle ear, one will discover a mass of pus usually rather fluid in consistency and apparently endless in amount if an attempt is made to swab it all out.

In the more chronic cases due to grass awns, incoordination is slight or not observed, and tenderness on palpation may be entirely absent, but the bulla is enlarged on the affected side. The discharge from the ear is of an intermittent nature.

HISTORY

The histories of the cases presented in our clinic indicate that a discharge from the auditory canal has existed either continu-

ously or intermittently for from three months to two or three years. Many remedies have usually been tried, some of them apparently successful for the time being; the ear clearing up, only to begin discharging again about the time the clinician begins to congratulate himself for having found "the treatment" for otitis.

Then there is the occasional case like two that occurred in our clinic last year. Both of them were of recent origin. Close examination revealed in the middle ear, past the location of the tympanum, the end of an awn which it was not possible to remove. Repeated efforts failed to get the offending foreign body. Both of these cases responded to the usual treatment of thoroughly cleansing with ether, followed by the use of olive oil. They apparently recovered, only to return for further treatment a few months later.

The reason that such a position may be taken by the foreign body is readily seen when one takes into consideration the anatomy of this region. The awn gradually works inward, punctures the tympanic membrane, and gains the cavity of the tympanum. It would naturally have a tendency to drop downward, thus falling into the large, single cavity of the bulla ossa which is ventral and slightly medial to, and in direct communication with, the tympanic cavity. The object is then out of a direct line with the auditory canal and may defy all efforts of the clinician to dislodge it.

The type of case just described is the most frequent in our clinic. However, there has occurred otitis media associated with otitis externa from ear-mite infestation with a perforated ear drum. This type is usually more acute and shows at times more or less incoordination followed by copious discharges of an offensive material which gives temporary relief, followed by repetition of the cycle at various intervals. After it is apparent that treatment through the auditory canal is of no avail, the operation here described is indicated in all of the above conditions as well as for acutely infected inflammatory conditions which may have occurred without perforation of the drum.

Surgical Anatomy

The bulla ossea is relatively large in the dog and constitutes the most ventral part of the petrous temporal bone (figs. 6 and 7). It consists of a shell of bone surrounding a single cavity or excavation which communicates so freely with the cavity of

the tympanum as to be considered a diverticulum of the latter. The shell of bone is relatively thick laterally and anteriorly; but thin ventrally, posteriorly and medially (when diseased, the bone is thickened and appears somewhat spongy.) The cavity of the bulla ossea is situated ventral and slightly medial to the tympanic cavity. It is lined by a modified mucous membrane similar to, and continuous with, that of the tympanic cavity, which in turn is continuous with the mucous membrane of the pharynx by means of the Eustachian tubes.

The bulla ossea is usually readily palpated in spite of its depth. This is possible because of the relatively loose nature of the fasciae and areolar tissues of the dog. Just posterior to the bulla is the short paramastoid process. The occipito-mandibularis (digastric) muscle arises from the paramastoid process and extends forward and downward over the bulla ossea. Anterolateral to the bulla is the prominent postglenoid process. The hyoid bone passes over the lateral side of the bulla ossea and has a cartilaginous attachment to the temporal bone lateral and anterior to the base of the paramastoid process (fig. 6). The hyoid bone serves as a landmark in locating the bulla ossea when operating. The topography of the vessels and the nerves concerned will be discussed under technic.

Our first attempt at bulla-osteotomy was from a lateral approach, but this was soon discarded as impractical, if not impossible, for the following reasons: (1) Proper drainage could not be provided. (2) It was very difficult to avoid the large vessels concerned. (3) The bulla ossea is thick laterally but over its ventro-medial part it consists of a very thin plate of bone. This thin part could not be reached from a lateral approach and, at the angle necessary to work, it was very difficult to penetrate the bone.

On the other hand, the loose areolar tissue just lateral to the larynx and pharynx affords a ready means of approach from below. The ventral approach also affords a perfect drainage, the vessels and nerves are readily moved out of the operative field, and the penetrating instrument can be placed squarely upon the bone at its thinnest part.

INSTRUMENTS

Each individual will vary somewhat in his choice of instruments for a given operation. We lay no claim to having exhausted the field relative to the type of instruments which might

prove advantageous in performing this operation, but will mention the instruments that we have found indispensable. The usual scalpel for skin incision, hemostats, sponges, suture and needles are, of course, necessary. On account of the vital structures located in, or near, the field of operation, it is necessary that much blunt dissection be done. While we have accomplished this blunt dissection with the handles of scalpels and the closed ends of rather pointed hemostats, we wish very emphatically to discourage the use of such instruments. They destroy or bruise too much tissue. One of the writers (McCoy) has made two "blunt dissectors." These instruments are about seven and one-half inches long over all. The blade is about three and one-half inches long and comes to a blunt, slightly curved end, having a width of about one-eighth inch, and a thickness of about one-sixteenth inch. We have found these instruments very convenient and practically indispensable in the course of the operation. A wound retractor is essential. The Mayo retractor, designed for the roaring operation, is very satisfactory. Bone instruments consist of a small, sharp-pointed trocar or similar instrument to make the "seat" and a "plain tooth drill" to enlarge the opening to the desired size. Have on hand a probe or applicators to swab out the cavity of the bulla; alligator forceps to remove foreign material and for use in packing; a five- or ten-cc hypodermic syringe fitted with a Quitman probe-pointed teat canula (useful for irrigating purposes); some sterile gauze and mercurochrome for packing. With this material properly sterilized and complete anesthesia established, one is ready to proceed with the operation.

Technic

After the proper preparation of the region, the dog is placed squarely upon its back with the head moderately extended. The point of incision is medial to the mandibular salivary gland and just lateral to the external maxillary vein (fig. 1). The mandibular salivary gland can be readily located by palpation, just posterior to the ramus of the mandible, and the external maxillary vein can be raised and seen through the thin, shaved skin of the region lying lateral to the larynx. The skin incision should be ample, carried from the mandibular space backward to the side of the larynx. This exposes the thin, but complete, cutaneous muscle which should be incised the same as the skin. The mandibular salivary gland will now be exposed, covered by

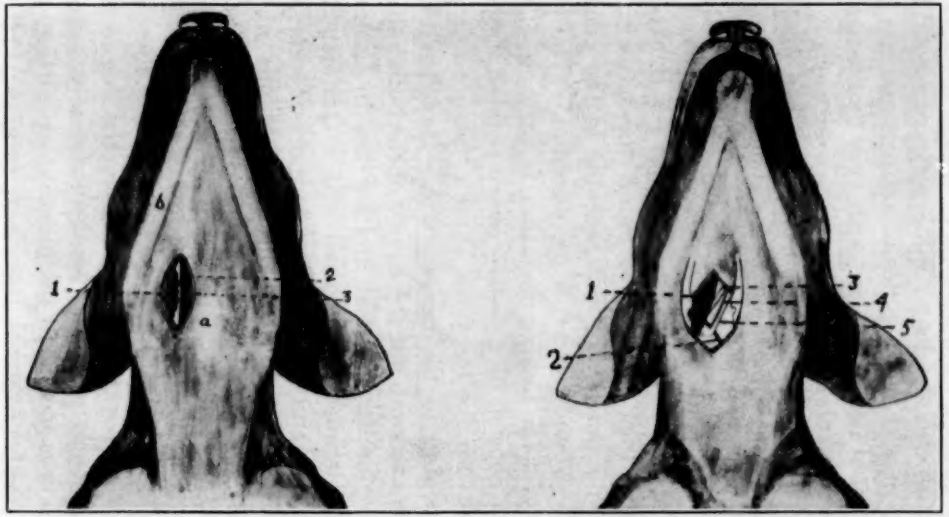


FIG. 1 (left).—The point at which the skin incision is made can readily be seen from this figure. a, position of the larynx; b, the ramus of the mandible; 1, manidublar salivary gland; 2, external maxillary vein; 3, cutaneous muscle incised the same as the skin.

FIG. 2 (right).—The soft areolar tissue lateral to the larynx and pharynx has been divided by blunt dissection and retractors inserted to show the next step in the operation. 1, occipito-mandibularis muscle; 2, external carotid artery; 3, hyoid bone (the muscle covering this bone is necessarily not shown so that the position of the bone may be seen); 4, lingual artery; 5, hypoglossal nerve.

a special fascia or capsule, and the external maxillary vein is seen medially. From this stage on, blunt dissection must be carried out.

The capsule surrounding the salivary gland should not be opened, but tear the loose areolar tissue between the gland and vein, proceeding slightly medially and forward. Make no effort to follow the extrinsic laryngeal muscles because if these muscles are followed, one is almost certain to injure the descending radical of the hypoglossal nerve, a small nerve lying close to the above muscles. This nerve sends branches to the thyrohyoideus and sterno-thyro-hyoideus muscles and joins the first cervical nerve. After an opening of sufficient size has been made, locate the hyoid bone with the finger (fig. 2). The hyoid bone is not really exposed, as shown in the figure, but is covered by the stylo-glossus and the stylohyoideus muscles. However, it can be palpated readily and serves as a guide to direct the operator to the exact point where the bulla ossea is to be opened. The large lingual artery lies just posterior to the hyoid bone, and just posterior to the artery is seen the hypoglossal nerve. The nerve and artery are not covered by muscle but are laid bare by the blunt dissection. Lateral to the structures mentioned is the occipito-mandibularis muscle.

The retractors should now be used, the nerve being carefully dissected up and placed laterally against the occipito-mandibularis muscle, and the retractor inserted to force the nerve and muscle laterally and the lingual artery and hyoid bone medially. The external carotid artery is now found directly in the field of operation. It is best forced under the lateral wing of the retractor. Under no condition should this artery be injured, as it furnishes the principal blood-supply to the head. The lingual and external carotid arteries diverge markedly after the lingual leaves the latter, hence there is little danger of injuring either by placing the retractor between the two. However, if an attempt is made to force the external carotid medially, there is danger of rupturing the external maxillary or the radicals to the occipito-mandibularis muscle.

The bulla can now be located with one's finger as it projects medially under the occipito-mandibularis muscle (fig. 3). The muscle, when well developed, covers the greater portion of the bulla, making it necessary to nick it or cut a portion out of it medially (fig. 4). When the muscle is not so well developed it is readily removed from the seat of operation without the necessity

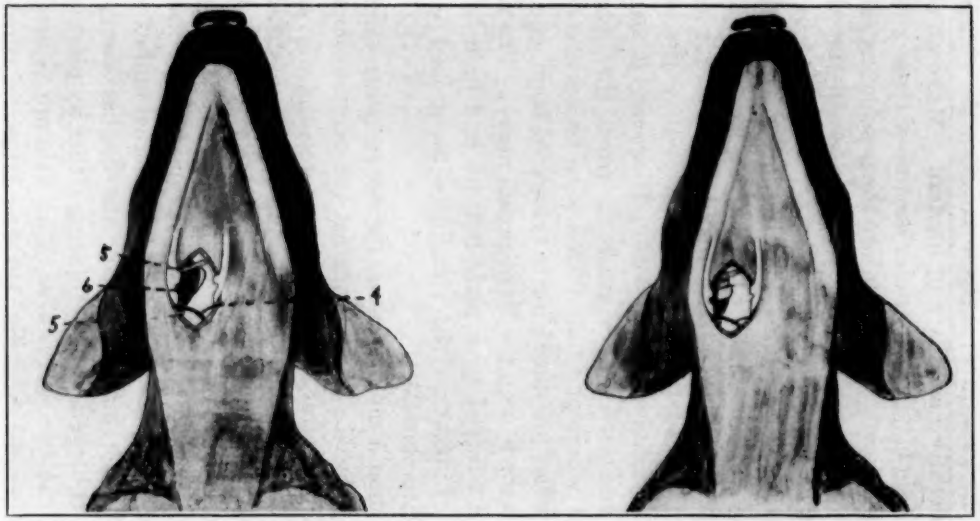


FIG. 3 (left).—The retractors have been placed between the hyoid bone and the occipito-mandibularis muscle, forcing the lingual artery and hyoid bone medially; the hypoglossal nerve, external carotid artery, and occipito-mandibularis muscle laterally. 4, lingual artery; 5, hypoglossal nerve; 6, occipito-mandibularis muscle near its origin. This muscle is not as large as represented, except in heavily muscled dogs. The bulla ossea can be seen projecting medially from the border of the muscle but is not labeled.

FIG. 4 (right).—The same as figure 3 except a small section has been removed from the occipito-mandibularis muscle showing more of the bulla ossea. A small vein is seen running transversely across the bulla. Sometimes a small artery also is encountered in this region.

of injury. The muscle should never be completely divided. The hyoid bone should be located and traced until it meets the skull. A point just posterior to this is the proper place to open the bone. A small vein is usually found passing transversely over the anterior part of the operative field. A small artery also may be found having an oblique or longitudinal course. These should be removed from the field without injury and the periosteum scraped from the bone preparatory to penetration. A small, sharp-pointed drill or trocar is now used to make a small opening or "seat" in the bone preparatory to using the larger instrument. This is done as the surface of the bone is rounding and the larger instrument may slip to one side, which might easily prove fatal, due to cutting one of the large blood-vessels of the region. The larger drill may then be placed in the "seat" and the opening enlarged to any size desired. It is necessary to make the opening large enough to afford ample drainage but the entire floor of the bulla need not be removed.

"Cardinal Points" of the Operation

The aim of the surgeon should be to perform as nearly a bloodless operation as possible. The drawing of any quantity of blood interferes markedly with the identification of the vital structures involved and would also fill the tympanic cavity with blood immediately following the opening of the bulla.

Bulla-osteotomy is not performed without danger, as may be seen by the vital vessels and nerves lying directly in the field of operation. However, we have performed a dozen operations without injury to either, except in one case as noted below. The hypoglossal nerve must be handled very carefully as injury results in paralysis of the muscles of the tongue, and improper deglutition.

In order to determine how serious injury of the hypoglossal nerve might be, both nerves were sectioned in an experiment dog, below the point where the descending radicle of the nerve is given off. This dog was not able to advance the tongue from the mouth but could withdraw it if the tongue were grasped and drawn out through the lips. For the first week it seemed unable to swallow either food or water unless placed on the root of the tongue. It would frequently cough when swallowing liquids. After this, the dog learned to eat solid foods, but made no effort to masticate them. It would take liquids by a biting, chopping movement. The dog was destroyed at the end of one month, showing no apparent improvement in its ability to eat or drink.

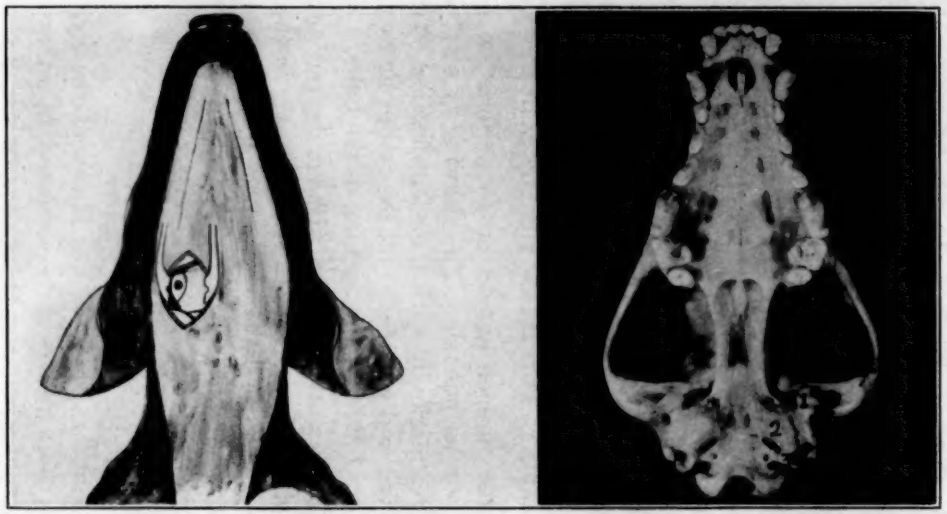


FIG. 5 (left).—The muscle has been pushed back, the periosteum removed from the bone and the bull aossea opened.

FIG. 6 (right).—Ventral view of a dog skull (Scottish terrier) to show the position of tne bulla ossea. 1, Po.tglenoid process; 2, bulla ossea; 3, paiamastoid process. The dotted line indicates the position of the cartilaginous prolongation of the hyoid bone in relation to the bulla ossea.

In another dog the descending radicles only of the hypoglossal nerve were cut. This dog controlled its tongue normally, and deglutition appeared normal except when large portions of meat were swallowed without proper mastication. In this case the dog would choke and the meat would be returned to the oral cavity. One experiment dog, in which double bulla-osteotomy

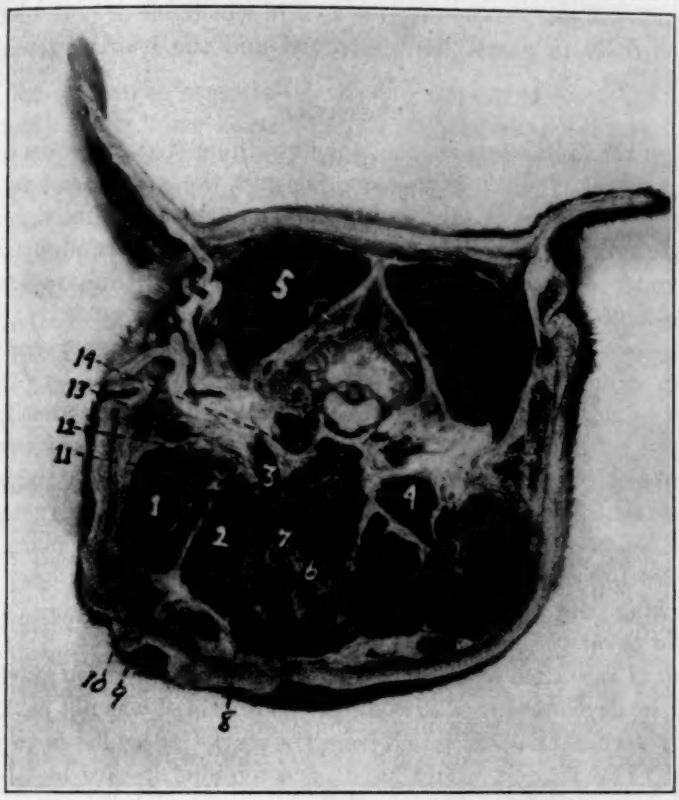


FIG. 7.—Trans-section of the head of an English bulldog through the ears. The structures seen ventral to the middle ear are slightly anterior to the operative field. 1, masseter muscle; 2, occipito-mandibularis muscle; 3, styloglossus muscle; 4, pterygoideus medialis muscle; 5, temporalis muscle; 6, posterior part of oral cavity; 7, tonsil, 8, hypoglossal nerve; 9, external maxillary vein; 10, mandibular lymph-nodes, also seen medial to 9 but not labeled; 11, external maxillary artery, 12, internal maxillary vein; 13, internal maxillary artery; 14, cavity of the tympanum. The cavity of the bulla ossea is seen communicating freely with the tympanic cavity. Above the dotted line can be seen the tympanum and the manubrium of the malleus attached to it.

On the opposite side a portion of the ear-drum has been cut but the ossicle is preserved. A portion of the external auditory canal also is seen. Note the angle of the ear-drum with relation to the long axis of the auditory canal.

was performed, showed the same symptoms. In this animal, however, large instruments such as hemostats and the handles of scalpels were used for blunt dissection and no effort was made to avoid the nerve in question. The symptom seen in these two dogs is, in all probability, due to injury to the nerve supply of the thyro-hyoideus muscle.

Great care should always be exercised not to injure the ossicles. These can be readily seen through the opening in the bulla with proper illumination. It also should be remembered that the manubrium of the malleus extends downward over the middle of the tympanum (fig. 7). Destruction of the ossicles results in deafness. The tympanum may be destroyed and the animal continue to hear. Many dogs are seen in which one or both drums are partially or completely destroyed and the hearing remains fairly good.

TREATMENT

After the bulla ossea is open, the next thing that confronts us is the removal of all foreign material and the establishment of drainage. Obviously, the ear drum is partly or wholly gone. Taking advantage of this fact, we have irrigated through from the outer ear with the long teat-canula and thus removed accumulations in the tympanic cavity. Then the cavity of the bulla ossea and wound area is swabbed dry, being very careful not to injure the ossicles.

To establish drainage a gauze pack is used, saturated with a solution of mercurochrome or other non-irritating antiseptic, the object being to establish a fistulous tract through the soft tissues so these tissues will not heal before the bony structures have had time to repair. This pack should be left in place for at least forty-eight hours. The wound should be kept open by repacking and curettage until it has stopped draining. This should never be less than three weeks.

Out of the dozen operations performed, one healed too quickly, with the development of an abscess. This animal was not packed, but treated as an open wound from the start. It healed in twelve days. This sequela would have been avoided if care had been used to keep the wound open for a longer period.

On some of our experiment dogs, we have purposely destroyed both ear drums and irrigated through the external auditory canal. After the wound healed, the hearing of these dogs was still good. Hence the conclusion is drawn that unless changes leading to deafness have taken place prior to operation, the operation itself, if properly done, will not lead to deafness.

In all the cases operated complete recoveries followed, with no complications except the one abscess and the one case with difficult deglutition already mentioned. With but one exception, these dogs were bright, active, and ready to eat the morning after the operation. They submitted to frequent treatment of the wound without any unusual objection.

PURCHASED VS. HOME-GROWN REPLACEMENTS: THEIR RELATIVE VALUES IN BUILDING UP HERDS FREE FROM BRUCELLA INFECTION*

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The principles essential to the building-up of herds free from Brucella infections have been demonstrated and proven. In general, the procedure has been that successfully employed in the control of other contagious or infectious diseases of cattle, namely, the detection of infected animals, the elimination of these, disinfection of premises, quarantine measures, sanitary and hygienic precautions, and replacement of infected cattle with those believed to be free from disease. Without reviewing the several plans that have been formulated and operated with varying degrees of success, it is obvious that the best method for controlling Brucella infections is that which requires the most stringent precautions to eliminate all infection from a herd and to prevent introduction of new infection.

Although it is recognized that the more exacting plans have their merits in promising earlier and fuller success, such plans may be restricted in their general application. There are a very considerable number of dairy herds in which the adoption of absolute measures for the control of Bang disease is physically impossible, economically impractical, or both. Within the past three or four years, many herds producing the higher grades of milk have had to initiate control work on a modified basis, contending with the all-too-evident weaknesses inherent in compromise measures.

Some of the major handicaps of the compromise plan are: first, that temporary retention or physical separation of reactors have had to act as substitutes for immediate elimination or complete isolation; second, that replacement cows and herd additions have, of necessity, been purchased in advanced stages of gestation; third, that purchases have had to be made on the basis of one or two blood-tests only and from herds known to be infected. Undesirable as these things may be, yet many dairy herds that are in

*Read before the Northeastern States Conference on Infectious Abortion, Ithaca, May 23, 1930.

the stage of transition from an infected status to a prospective Bang-free status must face just such problems.

In our own experience, the big problem has been the replacement or herd-addition cow. In a previous report,[1] some data were given relative to certain phases of buying replacement cows subject to the agglutination test for Brucella infections. It was shown that, by employing a carefully controlled system of check-testing, it is possible to buy considerable numbers of dairy cattle with a fair degree of satisfaction insofar as immediate results are concerned. The present report deals with the permanency of beneficial results as observed in purchased replacements after

TABLE I—*1928 purchases. 287 purchased blood-negative replacements and their subsequent behavior to agglutination tests*

LOAD	NEGATIVE COWS	POSITIVE TEST AFTER CALVING		BEHAVIOR AFTER ADDITION TO HERD			
				MONTHS IN HERD	NEGATIVE COWS	POSITIVE TEST	
		No.	%			No.	%
1	26	2	7.7	23	24	4	16.6
2	29	0	0.0	23	29	5	17.5
3	26	0	0.0	22	26	7	26.9
4	29	0	0.0	22	29	4	13.8
5	31	8	25.8	22	23	9	39.5
6	28	0	0.0	21	28	5	17.8
7	30	0	0.0	21	30	3	10.0
8	33	1	3.3	20	32	7	21.9
16	31	1	3.2	20	30	8	26.6
18	24	4	16.6	20	20	8	40.0
Totals	287	16			271	60	
Averages			5.6	21.4			22.1

being in the herd for periods up to about two years, compared with similar observations on home-grown heifers. These observations are summarized in the accompanying tables.

In presenting the data, only three factors have been taken into consideration for the purposes of this report. These factors are: the numbers of purchased cattle and home-grown heifers, entirely negative to agglutination tests when received, which reacted after calving; the subsequent behavior of the cows and heifers, still negative after calving, to repeated blood-tests after entrance to the herd; and, the time at which cows or heifers reacted to tests after being added to the herd.

Purchased Replacements

Table I concerns ten loads of replacement cows purchased in 1928, showing the number of cows with negative blood-tests in each load, and the number and percentage of these that reacted positively when tested about twenty days after calving. The "Behavior after Addition" data relate to the same cattle, except that those reacting to the first test after calving have been excluded, and show the length of time each load has been in the herd and the number of positive cows encountered on subsequent retests.

Of 287 negative cows purchased in 1928, 16 (5.6 per cent) reacted to agglutination tests after calving. This figure is somewhat above that which we have come to regard as the normal expectancy. Of the 271 cows still negative after calving, 60 (22.1

Table II—*1928 purchases. Time at which 60 blood-negative replacements reacted to agglutination tests after addition to herd*

Months in Herd	Positive Test		Months in Herd	Positive Test	
	No.	%		No.	%
1	0	0.0	11	3	5.0
2	1	1.6	12	6	10.0
3	1	1.6	13	4	6.6
4	0	0.0	14	1	1.6
5	9	15.0	15	0	0.0
6	4	6.6	16	4	6.6
7	9	15.0	17	0	0.0
8	9	15.0	18	0	0.0
9	6	10.0	19	0	0.0
10	2	3.3	20	1	1.6

Of the 60 cows reacting within 2 to 20 months after entrance to herd: 2 (3.2 per cent) reacted within 2 to 4 mos.; 31 (51.6 per cent) within 5 to 8 mos.; 17 (28.3 per cent) within 9 to 12 mos.; 9 (14.8 per cent) within 13 to 16 mos.; 1 (1.6 per cent) within 17 to 20 mos.

per cent) eventually gave positive tests after being in the herd for an average of 21.4 months. This figure also is higher than the expected rate.

Table II shows the time in months after the cows were added to the herd, at which the 60 animals noted in the seventh column of table I reacted to retests. More than half of these reacted within five to eight months, and nearly eighty per cent within twelve months after entrance to the herd.

Table III is similar to table I, covering replacement cows purchased in 1929. Of 535 such cows with negative blood-tests, 19 (3.5 per cent) gave positive results when retested about twenty days after calving. This figure is about that which we believe is the normal expectancy for reactions in pregnant cows purchased

subject to test, out of infected herds. Of the 516 cows still negative after calving in 1929, 58 (11.2 per cent) subsequently reacted to retests over an average period in the herd of 10.8 months. This percentage of reactions is half that for the 1928 group, but the

TABLE III—*1929 purchases. 535 purchased blood-negative replacements and their subsequent behavior to agglutination tests*

LOAD	NEGATIVE COWS	POSITIVE TEST AFTER CALVING		BEHAVIOR AFTER ADDITION TO HERD			
				MONTHS IN HERD	NEGATIVE COWS	POSITIVE TEST	
		No.	%			No.	%
20	17	2	11.7	14	15	1	6.6
21	29	0	0.0	14	29	1	3.4
22	27	1	3.6	13	26	1	3.8
23	22	1	4.3	13	21	1	4.8
24	32	0	0.0	13	32	4	12.5
25	28	3	10.7	12	25	9	30.6
26	23	0	0.0	11	23	7	30.4
27	27	0	0.0	11	27	3	11.1
28	55	2	3.6	11	53	5	8.4
29	21	0	0.0	10	21	3	13.3
30	25	2	8.0	10	23	2	8.7
31	27	0	0.0	10	27	3	11.1
32	42	2	4.8	10	40	3	7.5
33	29	0	0.0	9	29	3	10.3
34	15	0	0.0	9	15	1	6.6
35	21	0	0.0	9	21	0	0.0
36	16	1	6.2	9	15	4	26.6
37	56	4	7.1	9	52	6	11.5
38	23	1	4.3	9	22	1	4.5
Totals	535	19			516	58	
Averages			3.5	10.8			11.2

TABLE IV—*1929 purchases. Time at which 58 blood-negative replacements reacted to agglutination tests after addition to herd*

MONTHS IN HERD	POSITIVE TEST		MONTHS IN HERD	POSITIVE TEST	
	No.	%		No.	%
1	0	0.0	7	12	20.6
2	1	1.7	8	7	12.0
3	3	5.2	9	3	5.2
4	6	10.3	10	2	3.5
5	11	19.0	11	1	1.7
6	12	20.6			

Of the 58 cows reacting within 2 to 11 months after entrance to herd: 10 (17.2 per cent) reacted with 2 to 4 mos.; 42 (72.3 per cent) within 5 to 8 mos.; 6 (10.3 per cent) within 8 to 11 mos.

time in the herd for the 1929 purchases is also half of that for the 1928 group. In spite of this, the "critical period" for the 1929 cows has been covered, in our belief, since tables III and IV show

that the majority of replacement cows that are apt to succumb to retests do so within a period of five to twelve months after entrance to the herd.

Home-Grown Heifers

Table V is similar to tables I and III but give data regarding 46 home-grown heifers added to the herd in 1928. This group

Table V—*1928 Home-grown heifers. 46 home-grown blood-negative replacements and their subsequent behavior to agglutination tests*

Group	Negative Heifers	Positive Test After Calving		Behavior After Addition to Herd			
				Months in Herd	Negative Heifers	Positive Test	
		No.	%			No.	%
1	8	4	50.0	20	4	3	75.0
2	17	4	23.5	19	13	4	30.7
3	10	1	10.0	18	9	2	22.2
4	11	1	9.9	17	10	2	20.0
Totals Averages	46	10	21.7	18.5	36	11	30.5

Table VI.—*1929 home-grown heifers. 150 home-grown blood-negative replacements and their subsequent behavior to agglutination tests*

Group	Negative Heifers	Positive Test After Calving		Behavior After Addition to Herd			
				Months in Herd	Negative Heifers	Positive Test	
		No.	%			No.	%
5	17	1	5.9	16	16	2	12.5
6	10	0	0.0	15	10	1	10.0
7	13	0	0.0	14	13	0	0.0
8	12	0	0.0	13	12	2	16.6
9	26	0	0.0	12	26	2	7.7
10	4	1	25.0	11	3	0	0.0
11	26	0	0.0	10	26	0	0.0
12	7	0	0.0	9	7	0	0.0
13	14	0	0.0	8	14	0	0.0
14	8	0	0.0	7	8	0	0.0
15	9	0	0.0	6	9	0	0.0
16	4	0	0.0	5	4	0	0.0
Totals Averages	150	2	1.3	10.5	148	7	4.7

makes the poorest showing of any, partly due to the fact that control of Brucella infections on our young-stock units was not begun until the middle of 1928. On the initial test of these units, considerable infection was encountered and several months were

required to weed out infected animals. In addition, it is generally believed that negative heifers added to a herd in the cleaning-up process are more susceptible to the infection still present than are blood-negative, mature, native dairy cows. The data in table V are not particularly significant but are inserted for comparative purposes.

Table VI gives some indication of the results that may be expected after heifer-units have been under control and after the conditions in the producing herd to which heifers are added have been progressively improved. Of 150 heifers with negative blood-tests added to the herd in 1929, only two (1.3 per cent) reacted to retests twenty days after calving. Of the remaining 148 heifers, seven (4.7 per cent) reacted to retests after being in the herd for an average of 10.5 months. Both figures for reactions are less than half those obtained with the 1929 purchased replacements and may be indicative of the better results that may be obtained with home-grown replacements.

Again, the "critical period" for heifers added to the herd coincides, as might be expected, with that for purchased cows. The time periods after entrance to the herd at which negative heifers reacted to retests have not been tabulated but analysis of the figures shows that 95 per cent of the reactions occurred within twelve months.

Discussion and Summary

In judging the results that are presented in this report, it is probable that the experience in small dairy herds in many instances will not correspond with our own. The problem of controlling Brucella infections in large herds is much more complex than in small ones. Such advantages as the large herd may have in veterinary supervision, segregation and quarantine facilities and the like, are outweighed by the very multiplicity of details incident to supervising large numbers of animals during the dry and calving periods, which are of special importance in the control of contagious abortion. We do know that our experience corresponds very closely with that in other herds of fair size where considerable numbers of replacements have been made under similar conditions of blood-testing and segregation. It is, of course, well known that cows with negative blood-tests added either to infected herds or to herds in the cleaning-up process are more susceptible to whatever infection is present than are the native herd cows with negative tests. However, an unexpected

result is that first-calf heifers, raised from our own herd cows, which ordinarily are fertile soil for infection, are apparently more resistant than are mature dairy cows imported from other herds.

In obtaining replacements for dairy herds that are in the process of developing a Bang-free status, two methods are discussed: the purchase of mature cows and the raising of heifers grown on units where Brucella infections are being controlled.

When pregnant cows are purchased subject to agglutination tests from herds that are infected or of unknown status, a certain proportion will react soon after calving that were negative before. The normal expectancy for this condition is about 4 per cent. For 287 negative cows purchased in 1928, the figure is 5.6 per cent; and for 535 negative cows purchased in 1929, the figure is 3.5 per cent.

Home-grown heifers raised on units where Bang infection has been practically eliminated show less than half the above percentage of reactions when tested soon after calving. For 150 such heifers added to the herd in 1929, the figure is 1.3 per cent.

In addition to the positive reactions detected in negative replacements when retested twenty days after calving, an additional number of reactors may be found at various intervals after the animals have been added to a herd in the cleaning-up process.

The critical period during which most of these reactions occur is from five to twelve months. After this time very few negative replacement cows will react. This would indicate that the susceptible animals have largely succumbed and the remainder must be considered at least resistant.

In 271 replacements purchased in 1928, 60 (22.1 per cent) reacted over a period averaging 21.4 months; of 516 such cows in 1929, 58 (11.2 per cent) reacted over a period averaging 10.8 months; in 148 home-grown heifers added in 1929, 4.7 per cent reacted over a period of 10.5 months.

There is a marked balance in favor of the home-grown heifers, both with respect to reactions after calving and to subsequent retests while in the herd.

REFERENCE

[1]Hardenbergh, J. G.: Buying replacement cows free from *Brucella abortus* infection. Jour. A. V. M. A., lxxvi (1930), n. s. 29 (3), pp. 298-307.

The Council on Pharmacy and Chemistry of the American Medical Association recently completed twenty-five years of useful service to physicians and pharmacists.

THE INTERAGGLUTINABILITY OF MEMBERS OF THE BRUCELLA AND PASTEURELLA GENERA

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During the past two years, reports of finding Brucella infection in various animals other than cattle, goats and humans have appeared. Its occurrence in fistula of the horse (Fitch, Delez and Boyd[1]), in chickens (Emmel and Huddleson[2]), in the turkey, pigeon, pheasant, duck and goose (Emmel[3]), in sheep (Bruce[4]), and in hygroma of the knee of cattle (Boyd, Delez and Fitch[5]), has been reported. The writer does not question the authenticity of the above-reported findings, but it has occurred to him that perhaps other microörganisms might produce antibodies in the animals reported, that might cross-agglutinate with *Brucella abortus* antigen and thus cause confusion. Knowing the close

TABLE I—*Cross-agglutination studies with positive abortus cow serum by members of Brucella, Pasteurella and Pfeiferella groups*

ANTIGEN	CONTROL	SERUM DILUTIONS						
		1-20	1-40	1-80	1-160	1-320	1-640	1-1280	1-2560
Br. abortus	—	++++	++++	++++	++++	++++	++	+	—
Br. suis	—	++++	++++	++++	++++	++++	+++	—	—
Past. avicida	—	++++	++++	+++	++	+	—	—	—
Past. boviseptica	—	—	+	++++	++++	++++	+	—	—
Pfeif. mallei	—	+++	+++	++	++	—	—	—	—

morphological and physiological relationship that exists between members of the Pasteurella, Pfeiferella and Brucella groups, members of these groups were selected for this preliminary study.

Accordingly, antigens of the members of these groups available in the laboratory were prepared and tested with positive *Brucella abortus* serum obtained from a cow naturally infected. The results are presented in table I. It will be observed that the agglutination obtained with *Pasteurella boviseptica* was equally as good as either the *Br. abortus* or *Br. suis* antigens. The *Past. avicida* antigen, although not agglutinating at so high a titre as the Brucella antigens, still gave a marked agglutination. *Pfeif.*

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mallei also produced a marked agglutination but of a still lower titre.*

It is interesting to note that, in the usual dilutions used in routine testing, namely, 1:50, 1:100 and 1:250, the *Past. avicida* and *Past. boviseptica* would give the same results as Brucella antigen.

The next point to determine was the activity of a Pasteurella serum on a Brucella antigen. Accordingly, rabbits were immunized with various strains of Pasteurella antigens. These immune sera were then tested for agglutinin content for Brucella antigens.

TABLE II—*Cross-agglutination studies with an immune Past. boviseptica rabbit serum by members of Pasteurella and Brucella groups*

ANTIGEN	CONTROL	SERUM DILUTIONS					
		1-50	1-100	1-200	1-400	1-800	1-1600
Past. boviseptica	—	++++	++++	++++	++++	++++	++++
Past. suiseptica	—	++	++	++	++	++	++
Br. abortus	—	+++	+++	+++	+++	+++	+++

TABLE III—*Cross-agglutination studies with immune Brucella abortus rabbit serum by members of Brucella, Pasteurella and Pfeiferella groups*

ANTIGEN	CONTROL	SERUM DILUTIONS					
		1-50	1-100	1-200	1-400	1-800	1-1600
Br. abortus	—	++++	++++	++++	++++	++++	++++
Past. avicida No. 1	—	++++	++++	++++	++++	++++	++++
Past. avicida No. 2	—	++++	++++	++++	++++	++++	++++
Past. boviseptica	—	++++	++++	++++	++++	++++	++++
Past. suiseptica	—	++++	++++	++++	++++	++++	++++
Pfeif. mallei	—	++++	++++	++	+	+	—

The results obtained with a *Past. boviseptica* serum are presented in table II. It will be observed that in this case, the agglutination results obtained with *Past. boviseptica* and *Br. abortus* were again practically identical. Distinct agglutination of *Past. suiseptica* was also obtained but was not so clear-cut as with the two other antigens. The data show the interagglutinability of the two groups, Brucella and Pasteurella. It would seem to indicate that the mere agglutination of a *Brucella abortus* antigen, by a suspected serum, in itself is not conclusive evidence of Brucella disease in unusual hosts.

*Further studies on *Pfeif. mallei*, together with members of the genus *Hemophilus* and *Past. tularense* and *Past. pestis*, are now in progress.

To check the results obtained with naturally infected *Br. abortus* cow serum, rabbits were immunized with cultures of *Br. abortus, Br. suis* and *Br. melitensis*. The immune sera obtained were then tested with Pasteurella antigens. The data for *Br. abortus* immune serum are presented in table III. It will be observed that reactions similar to those obtained with naturally infected positive serum occurred. In table IV, the results with *Br. suis* immune rabbit serum are presented. Here again similar results were obtained, further confirming the original findings.

Agglutinin-absorption studies now in progress seem to offer a means of separation and identification of the groups.

In this preliminary report, the possible significance of this interagglutinability will not be discussed. Suffice it to state that these findings, together with extensive occurrences of Pasteurella

TABLE IV—*Cross-agglutination studies with Brucella suis immune rabbit serum by members of the Brucella, Pasteurella and Pfeiferella groups*

ANTIGEN	CONTROL	SERUM DILUTIONS					
		1-50	1-100	1-200	1-400	1-800	1-1600
Br. suis No. 17	—	++++	++++	++++	++++	++++	++++
Br. suis No. 402	—	++++	++++	++++	++++	++	++
Past. boviseptica	—	—	+++	+++	+++	+++	+++
Br. abortus	—	—	—	++++	++++	++++	++++
Pfeif. mallei	—	++++	++++	++++	++++	++++	++++

in cattle, chickens and other animals, should caution workers who depend solely upon the agglutination test for the diagnosis of Brucella disease, including brucelliasis in man.

CONCLUSION

A preliminary report on the interagglutinability of members of the Brucella and Pasteurella groups is presented.

Brucella-immune sera were found to agglutinate Pasteurella and Pfeiferella antigens and Pasteurella-immune sera were found to agglutinate Brucella antigens.

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SIMPLE ATROPHY OF THE EYE-BALL

By J. F. BULLARD, *Lafayette, Indiana*

Department of Veterinary Science, Purdue University Agricultural Experiment Station

Subject: This case of simple atrophy of the eye-ball occurred in a Hereford cow.

History: The only history given was that this cow had shown a watery discharge from the affected eye for about three months.

Symptoms: A clinical examination revealed the following: The discharge from the eye was serous in character; the inflammation of the palpebral conjunctiva appeared to be of long standing; the cornea was clouded and opaque; and the eye-ball appeared as a solid dark mass.

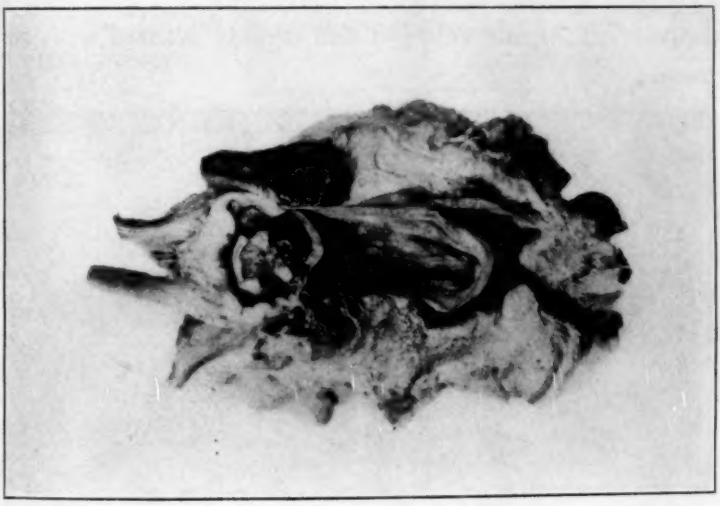


FIG 1. Longitudinal section of surgically removed tissue, showing the small solid eye-ball and the distended conjunctival sac. The membrana nictitans is immediately behind the lids, which appear at the right. The optic nerve is seen at the extreme left.

Treatment: The cow was placed on the operating-table, and the operative area cleaned, shaved, and washed with a disinfectant, after which a local anaesthetic was injected into the tissues at several different points. The eye-ball, lids and conjunctiva were removed. The ocular cavity was then cleaned, and packed with sterile gauze which had been previously immersed in ether and iodoform and the edges of the wound were brought together with mattress sutures. Part of the pack was removed the following day and the remainder in 48 hours, and the cavity filled with a paste composed of bismuth, iodoform and mineral oil. Healing was complete in about 10 days, when the stitches were removed.

Microscopic examination revealed a simple atrophy of the eye-ball.

A CASE OF LEUKEMIA IN THE FOWL

By M. W. Emmel* *and* H. J. Stafseth

Michigan Agricultural Experiment Station
East Lansing, Mich.

An owner brought a Barred Rock hen to the laboratory with a history that the bird had been affected for a week. The left leg was paralyzed but otherwise the bird seemed bright. Color and general condition were good. Upon autopsy it was found that the liver was slightly enlarged and densely spotted with small

*Resigned.

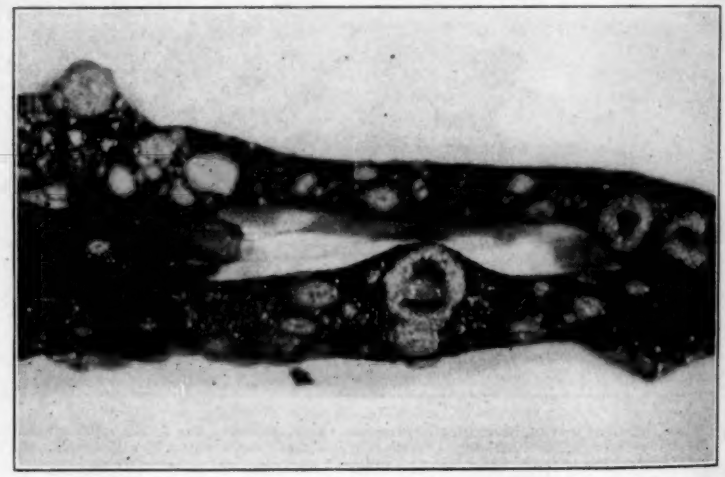


Fig. 1. An unusual intestinal lesion in a case of leukemia in the fowl.

yellowish-gray streaks and foci. It was rather friable but otherwise had the appearance of being leukemic, except perhaps it was darker in color than usually found in this disease. The spleen was twice normal size and many small grey foci were visible. The kidneys were intensely white, enlarged and closely resembled brain tissue in texture and appearance. The ovary was about 40 mm. in diameter, was a solid, globular, white, sarcomatous mass, and resembled the kidneys in texture and color.

A white, ovoid, tumor-like mass measuring 30 mm. x 17 mm. was found in the thoracic cavity attached to the under surface of the sternum in front of the apex of the heart. A second similar tumor-like mass occurred in the abdominal cavity, attached to the abdominal wall just to the left of the ovary but having no connection with the latter. These masses also resembled brain tissue in texture and color.

An unusual lesion, as pictured in figure 1, appeared in the intestines. Caudally, as far as the ceca, the mucosa was studded with numerous white pinpoint foci and with many larger ones, a few of which measured 13 mm. in diameter. A few of the larger foci appeared crater-like, depressed and slightly dark in the center, surrounded by an elevated, almost white ridge. Apparently these were foci of cellular infiltration but in no instance were the villi completely obliterated, as is so often the case where cellular infiltration occurs to any extent in the intestinal mucosa.

EPIDERMOID CANCERS ON THE FEET OF WILD BIRDS

By M. W. EMMEL,* *East Lansing, Mich.*

Michigan Agricultural Experiment Station

During the last few years, tumorous masses have been noted on the feet of wild birds caught for banding purposes. The specimens sent to our laboratory for study have been through the courtesy of Professor J. W. Stack, of the Zoology Department, who has been banding wild birds for a number of years and who states that this condition is quite prevalent, more especially in birds caught during the early spring.

Figure 1 shows a view of the right foot of a slate-colored Junco (*Junco hyemalis hyemalis*). The left foot was unaffected.

*Resigned.

The bulk of the tumor appeared as nodular masses in the region of the metatarsal-phalangeal articulation and involved the integument both dorsally and ventrally. In some instances the masses tended to extend into the region of the phalanges. On one extremity a nodule appeared on the dorsal surface of the last phalangeal articulation. The ventral surface of the masses was somewhat roughened but the integument appeared intact. The dorsal surface appeared scaly and at some points small necrotic fossae extended rather deeply into the nodular mass.

A microscopic study of the tumor revealed finger-like processes extending deep into the corium (fig. 2). Often these masses occurred singly but occasionally two or three such processes originated from a common source. Between the bases of these processes the epidermis was apparently normal. The epithelium

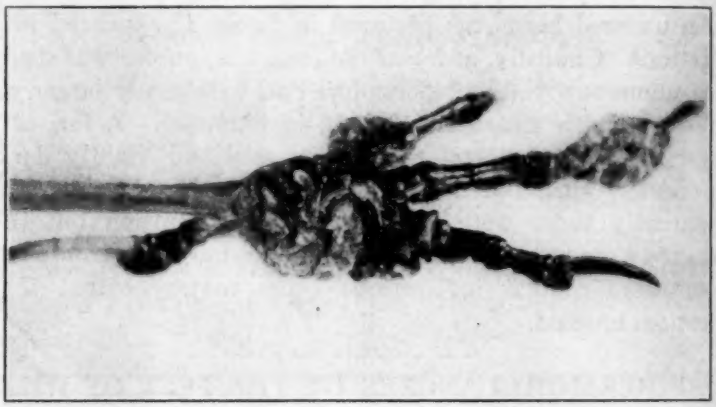


FIG. 1. Dorsal view of the right foot of a slate-colored Junco (*Junco hyemalis hyemalis*).

over the base of most of the smaller processes was normal but over the larger ones it was usually degenerative and necrotic and these changes tended to involve the central portion of the process often as deep as one-half its length. Below this point and still chiefly in the center of the mass, cells or groups of cells appeared necrotic. The remaining cells of the processes were hyaline in appearance. Only a few "epithelial pearls" occurred. The corium showed considerable inflammatory reaction at many points.

Since the above specimen was received, these tumors have been found on the feet of two other birds, one a slate-colored Junco and the other a bronze grackel (*Quiscalam quiscala aeneus*). The

gross and microscopic pathology were very similar to that described above.

Numerous birds are caught each year, usually early in the spring, which have these tumorous mass on their feet. Although

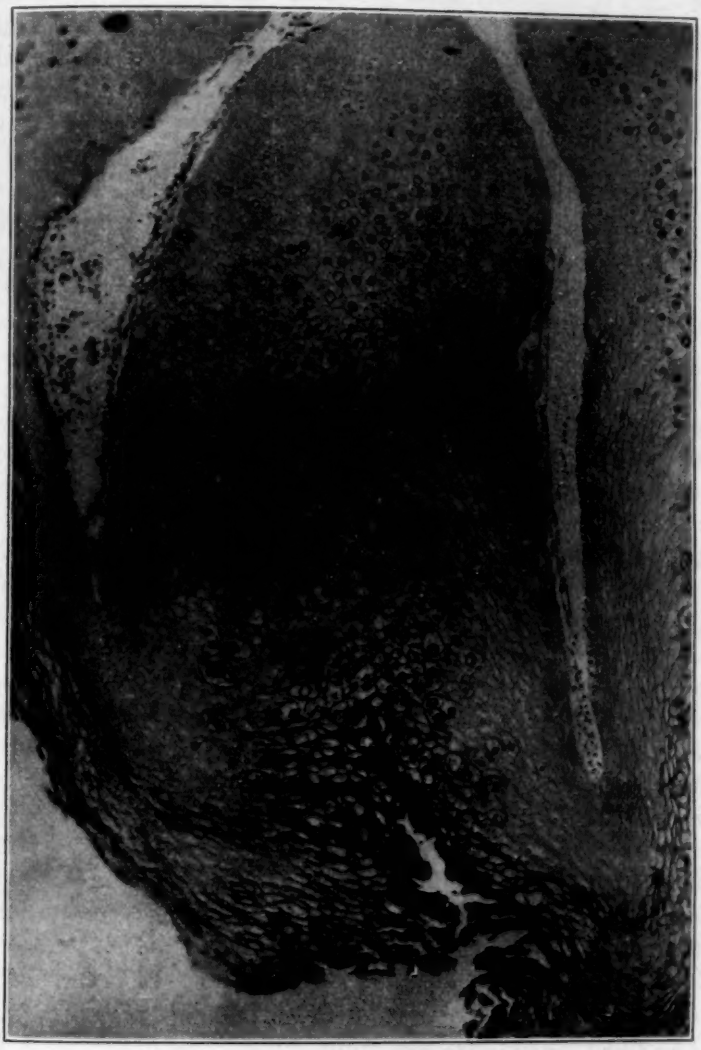


FIG. 2. Photomicrograph of an epidermoid cancer on the right foot of a slate-colored Junco (*Junco hyemalis hyemalis*). ca x400

no record of birds showing this pathologic process has been kept, Professor Stack states that a number of one-legged birds are caught each year. Thus the question arises as to whether there

may be any connection between epidermoid tumors on the feet and one-legged birds. Everything points to the non-malignancy of these tumors. As to their cause, it is suggested that birds returning northward early often encounter ice, sleet and snow and since it is usually more prevalent early in the spring it is highly possible that these neoplasms are a result of injury to the feet. As food becomes more easily obtained in the spring, the number of birds trapped decreases and consequently the number showing these tumors also is reduced.

EXPERIMENTS ON MAGNESIUM SULFATE AS A GENERAL ANESTHETIC

By O. W. JOHNSON, *Davenport, Wash.*

Having learned from a medical student that magnesium sulfate hypodermically, in 20 per cent solution is used by medical schools as a general anesthetic on animals being used for experiments. I decided to try it out for myself, and secured for the purpose a male cat. I was told that 10 cc of a 20 per cent solution would put a rabbit to sleep in a few minutes. Not knowing the dosage for a cat, I started out with 10 cc and waited for 15 minutes. The cat did not show any evidence of drowsiness, so I gave another 10 cc and waited another 15 minutes. This time the cat showed slight drowsiness. I then gave 20 cc. Within 15 minutes the cat was sound asleep and limp as a rag. After 10 minutes the breathing commenced to slow down and the heart action became slower and weaker. I injected 5 cc of a 20 per cent solution of calcium chlorid, hoping to revive him, but the cat died in a few minutes.

For a second experiment I secured 2 kittens, each weighing two pounds. To each one I gave hypodermically 10 cc of the 20 per cent solution of magnesium sulfate. Not getting any results, I injected 10 cc more in an hour. In two hours one kitten was sound asleep. On this I performed a laparotomy without any struggle or straining. I then gave 3 cc of a 20 per cent solution of calcium chlorid hypodermically. Within a few minutes the kitten was up on its feet.

The other kitten was not quite under the influence of the anesthetic, but I was able to castrate it without any restraint. This one received no calcium chlorid.

The following day I administered 20 cc of a 20 per cent solution of magnesium sulfate to each kitten. Within an hour one kitten was found dead. This was the one which received no calcium

chlorid the previous day. The other one did not show any effect except slight drowsiness.

To a 2½-pound kitten I gave 20 cc of magnesium sulfate solution. In an hour I gave 10 cc more. A half-hour later, I performed an ovariectomy without a struggle. I gave 3 cc of the calcium chlorid solution after the operation. Two hours later, when the kitten was not showing any signs of waking up, I gave 5 cc of calcium chlorid, and a half-hour later another 5 cc. The kitten failed to wake up, and died during the following night.

Not having read anything in veterinary literature on the use of magnesium sulfate as a general anesthetic in smaller animals, I do not know how familiar the members of the veterinary profession are with its use, or how widely it is used in small-animal hospitals or how carefully the dosage has been worked out.

From my limited experience I judge that it is safe and efficient in proper dosage, but that the margin of safety is quite narrow. It is also too slow in action where a quick anesthetic is required.

If some one has a larger experience with its use, I would like to have him publish his experience in the JOURNAL.

CALF-KILL NOT POISONOUS

By H. W. HERRIOTT, *Bureau of Animal Industry, and*

E. M. GRESS, *Bureau of Plant Industry, Department of Agriculture, Harrisburg, Pennsylvania*

In certain sections of Pennsylvania is found a shrub which is known among the farmers and other residents by the local name of "calf-kill," because it is thought to be poisonous to cattle and especially to calves. They say that every year some of the farmers lose cattle which have been pastured in fields in which this shrub grows.

In July, 1930, a specimen of this shrub was sent to the Bureau of Plant Industry, at Harrisburg, for identification and information on its poisonous qualities. The plant proved to be a shrub closely related to our ordinary sumac and poison ivy. Its scientific name is *Rhus trilobata* and its generally accepted common name, "ill-scented sumac," which is, indeed, quite appropriate, for its crushed leaves have a very strong and disagreeable odor.

Ill-scented sumac is not listed among the plants poisonous to live stock. Consequently, when the report was made, stating

that suspicion was placed upon this shrub, the pasture was searched for other poisonous plants that might be causing the trouble. None were found, and since the pasture contained a great many of the ill-scented sumac shrubs, we began to wonder whether it still might be the offender.

After consultation with the Bureau of Animal Industry, it was decided that the Bureau should undertake a feeding experiment at its laboratory and experiment farm, in order to determine whether or not the ill-scented sumac possessed any poisonous qualities.

A well-developed bull calf, about eight months old, was selected for the experiment. The calf had been given no feed for twenty-four hours previous to beginning the experiment. This was done so that he would be more likely to eat the ill-scented, and no doubt nasty-tasting, leaves and twigs. This assumption was correct, for although bran was added, the best he would do was to eat only eleven ounces. Seventeen ounces were then ground very fine by passing it through a meat-grinder. This ground material which consisted of leaves and young twigs was then shaken up with distilled water and, by the use of a drenching-bottle, was given to the animal. He did not seem to be particularly pleased with this operation, but after using nearly a gallon of distilled water we succeeded in getting it into his stomach, with but very little loss. With the eleven ounces that he had rather reluctantly eaten and the seventeen ounces poured into him, he had approximately twenty-eight ounces. This, it was felt, was a pretty fair dose, at least as much or more than an animal would likely eat from the pasture at one feeding.

We then awaited symptoms and results. The only noticeable effect was a rather copious watering of the eyes and more frequent urination. In a few hours this symptom disappeared and the animal appeared normal in every respect. His temperature was taken which showed no deviation from the normal.

Approximately two pounds and five ounces of the twigs and leaves were fed also to a fully-grown Ayrshire cow. The results were precisely the same as those with the eight-months-old calf.

Our conclusion, therefore, is that ill-scented sumac (*Rhus trilobata*) is not poisonous to cattle at single feedings in quantities that would likely be eaten by an animal in pasture. However, it does not prove that the shrub does not contain poisonous qualities that might be injurious if eaten over a long period of time.

RUPTURED RIGHT AURICLE IN HEART OF DOG

By GERRY B. SCHNELLE, *Boston, Mass.*

Angell Memorial Hospital

The accompanying photograph shows a ruptured right auricle in the heart of a six-year-old, male Sealyham terrier.

The dog was greatly excited at the time, chasing squirrels, and, needless to say, collapsed and died immediately.

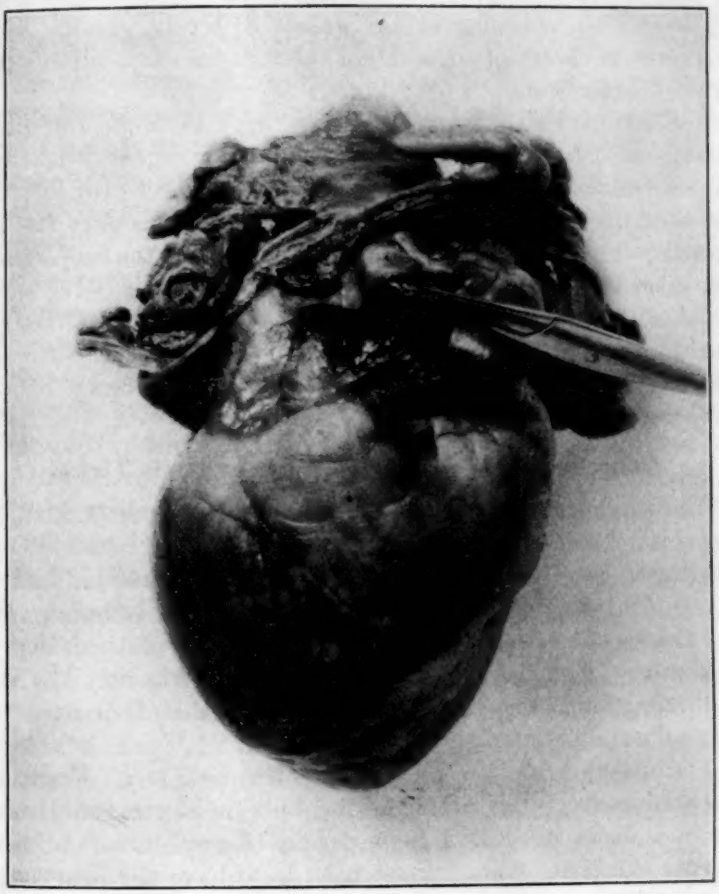


FIG. 1. Ruptured right auricle in heart of dog.

The photograph (three-quarters actual size) shows the character of the tear perfectly. The cardiac muscle seemed more friable than normal but there were no other macroscopic abnormalities.

I am unable to find any record of a case similiar to this, in veterinary literature available to me. Where cardiac rupture has occurred in the horse, according to Hutyra and Marek, it has been ventricular and the tear has been very small.

Retests Show Accuracy of Tuberculin

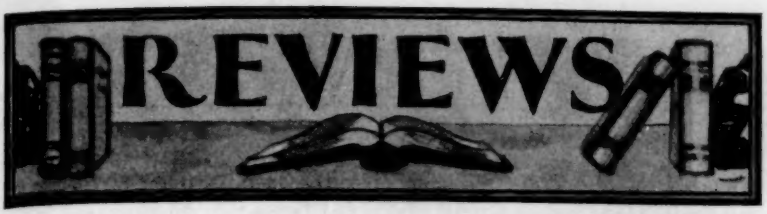
In four Wisconsin counties, where the cattle were tuberculin-tested three years ago and all reactors removed, a recent retest of 187,678 cattle disclosed only 189 reactors. This proportion of only about 1 in 1,000 evoked highly favorable comment by cattle-owners, according to Dr. James S. Healy, the B. A. I. inspector in charge of cooperative tuberculosis eradication work in the Badger State.

The counties included in the retest were Monroe, LaCrosse, Portage and Jackson. Notwithstanding the removal of reactors and the sale of a large number of cattle in the three-year interval between the tests, the number of cattle in each county has increased. The favorable attitude of the public and the cooperation extended by breeders greatly expedited the testing. The small number of reactors found three years after the original testing has impressed veterinary officers and herd-owners with the thoroughness of the previous work and the accuracy of the tuberculin test.

Cooperation Needed to Eradicate Cattle Ticks

The cattle tick remains a problem of prime importance to the agricultural welfare of the South, although the parasite has been eradicated from 10 of the 15 states originally quarantined for tick fever. The progress of tick eradication from its beginning, the life history of the tick, and details of eradication methods appear in Farmers' Bulletin 1057-F, "Cattle-Fever Ticks and Methods of Eradication," issued in revised form by the U. S. Department of Agriculture, in September.

The question of how to eradicate the tick is no longer an essential part of the problem, the bulletin shows, as present methods have reached a high degree of perfection and have proved effective. The greatest need is to have the hearty and conscientious cooperation of the people. Elimination of the tick is giving great impetus to beef production and dairying in the South. Farmers' Bulletin 1057-F may be obtained from the Office of Information, U. S. Department of Agriculture, Washington, D. C.



ALLGEMEINE PATHOLOGIE FUR TIERARZTE. (General Pathology for Veterinarians.) Theodor Kitt. 6th revised edition. xii + 570 pages, with 213 illustrations and 4 plates. Ferdinand Enke, Stuttgart, 1929.

The importance and purpose of this work may be made apparent by a few quotations from the introductory chapter, put here together at random: "General pathology, employing the science of etiology, attempts to ascertain why and how under certain conditions lesions are caused and disease symptoms produced . . . . . It inquires into the successive relation between origin, course and termination of abnormal function and unravels the natural laws by which definite reactions follow definite causes . . . . . It broadens out to become pathologic physiology and furnishes the scientific basis for the prevention and cure of diseases and thus becomes the backbone of prophylaxis and therapy . . . . . The layman and the empiric see only the external manifestations of disease, the pathologist is able to visualize the internal relationship and to act accordingly."

The usefulness of Prof. Kitt's book is attested by the subsequent translation of the first edition into English (in 1908) and by the continued demand for new editions of the original.. The rich and ripe experience of the writer and his erudition combine to make this present edition, which is practically entirely rewritten in the light of newer knowledge, a valuable source of reliable information not only for the veterinary student and practitioner but also for the medical research pathologist interested in comparative pathology. The conciseness of the definitions, the preciseness of expression and consequent lucidity of style, the clearness of type and the instructiveness of the illustrations make the perusal of this volume a delight even to the beginner in German.

F. V.

PUBLICATIONS RECEIVED

The Constituents of the Blood of Animals as Evidence of Intestinal Contribution to the Cause of Diseases of Obscure Origin. C. E. Hayden. Reprint from *Corn. Vet.*, xx (1930), 2, pp. 223-231.

The Production and Cure of Nutritional Anemia in Suckling Pigs. T. S. Hamilton, G. E. Hunt, H. H. Mitchell and W. E. Carroll. Reprint from *Jour. Agr. Res.*, xl (1930), 10, pp. 927-938.

A Method of Determining the Variations in the Vertebral Column of the Live Pig. A. M. Shaw. Reprint from *Sci. Agr.*, x (1930), 10, pp. 690-695.

Studies in Fascioliasis in Oregon Sheep and Goats. J. N. Shaw and B. T. Simms. (Sta. Bul. 266. Agr. Exp. Sta., Ore. State Agr. College, Corvallis, Ore., June, 1930.) Illustrated. pp. 24.

Hints on Wolf and Coyote Trapping. Stanley P. Young. (Leaflet 59. U. S. Dept. Agr., Washington, D. C., July, 1930.) Illustrated. pp. 8.

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Poultry Disease Laboratory at Farmingdale, Long Island, Report of the. J. M. Hendrickson and K. F. Hilbert. Reprint from *Ann. Rpt.* N.Y. State Vet. Coll., Cornell University, 1928-1929, pp. 49-53.

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Eradication of Pullorum Disease in Massachusetts, 1929-1930, Tenth Annual Report on. H. Van Roekel, K. L. Bullis and G. L. Dunlap. (Bul. 53. Mass. Agr. Exp. Sta., Amherst, Mass., 1930.) Illustrated. pp. 23.

Studies on Roup Vaccination in Chickens. Cecil Elder, F. J. Kohn and A. M. Lee. (Bul. 172. Univ. Wyo. Agr. Exp. Sta., Laramie, Wyo., 1930.) pp. 47.

Algunas Indicaciones sobre la Profilaxia de la Fiebre Carbunclosa. Dr. Jose Maria Quevedo. (Bul. 6. Facultad de Agronomia y Veterinaria, Universidad de Buenos Aires, Buenos Aires, 1930.) pp. 7.

Some Histological Studies of the Effects of Grubs Upon Animal Skin. Fred O'Flaherty and George D. McLaughlin. Reprint from *Jour. Amer. Leather Chemists Asso.*, xxv (1930), 7, pp. 266-270.

Relative Insecticidal Value of Commercial Grades of Pyrethrum. C. C. McDonnell, W. S. Abbott, W. M. Davidson, G. L. Keenan and O. A. Nelson. (Tech. Bul. 198. U. S. Dept. Agr., Washington, D. C., 1930.) pp. 9.

Why the Veterinary Profession? Pierre A. Fish. Reprint from *Corn. Vet.*, xx (1930), 3, pp. 301-304.

Game Laws for the Season 1930-31. (Farmers' Bul. 1647. U. S. Dept. Agr., Washington, D. C., 1930.) pp. 46.

Toxic Effect of St. Johnswort (*Hypericum Perforatum*) on Cattle and Sheep. C. Dwight Marsh and A. B. Clawson. (Tech. Bul. 202. U. S. Dept. Agr., Washington, D. C., 1930.) pp. 23.

Effect of the Electropure Process and of the Holding Method of Treating Milk Upon Bacterial Endospores. A. J. Gelpi, Jr. and E. D. Devereux. Reprint from *Jour. Dairy Sci.*, xiii (1930), 5, pp. 368-371.

Preparation of Acidophilus Milk from Milk Treated by Electricity. E. D. Devereux. Reprint from *Amer. Jour. Pub. Health*, xx (1930), 9, pp. 1009-1010.

Pennsylvania State Veterinary Medical Association, Extracts from the Proceedings of the 47th Annual Meeting of the. (Pittsburgh, Pa., October 24-25, 1929.)



TULAREMIA IN SHEEP IN NATURE.. R. R. Parker and J. S. Dade. Abst. Arch. Path., x (1930), 1, p. 135.

The proved occurrence of *B. tularense* in the tissues of sheep in nature opens the question of possibility of human infection from the handling of infected carcasses. Infection is definitely possible through the primary contamination of the hands with the tissues of crushed infected ticks, held in the wool or with thick excrement which is commonly present in large masses. The fingers might also become contaminated by contact with necrotic tissue which sometimes develops at the points where infected ticks have been attached. The chance that infected meat might reach the markets and be a source of danger to persons in slaughter-houses and packing-houses and to the consuming public seems less likely but cannot be altogether dismissed, especially if animals are slaughtered for immediate local consumption. Further and more carefully planned field and laboratory observations of tularemia in sheep caused by the wood tick are desired: (1) to determine the extent to which it is concerned in wood-tick-caused pathologic conditions, (2) to determine the geographical limits of the occurrence, which certainly are wider than indicated by present data, (3) to secure more detailed epidemiologic, symptomatologic and pathologic data and (4) to determine whether the meat of infected slaughtered sheep is a possible source of human infection.

THE ORAL TOXICITY OF EMETIN HYDROCHLORID AND CERTAIN RELATED COMPOUNDS IN RABBITS AND CATS. Hamilton H. Anderson and Chauncey V. Leake. Amer. Jour. Trop. Med., x (1930), 4, p. 249.

The minimum lethal dose of emetin hydrochlorid given orally in enteric-coated capsules is 15 to 20 mgm./kgm. for rabbits and cats. The minimum lethal dose of emetin bismuth iodid, emetin antimony iodid, auremetin and iso-amyl cephaelin phosphate is also given. Postmortem examination revealed degener-

ative injury to cardiac and smooth muscle and to liver and kidney following lethal doses of all of these drugs. Since the minimum lethal dose of emetin hydrochlorid by mouth is in the same range as that given subcutaneously, fairly complete absorption from the gut is indicated. Again, since the minimum lethal dose on single subcutaneous injection or on single dose by mouth is in the same range as when given in small divided doses daily, an extremely slow rate of elimination or detoxification of the drug is indicated.

Tooth Growth in Experimental Scurvy. Gilbert Dalldorf and Calia Zall. Jour. Exp. Med., lii (1930), 1, p. 57.

The incisor teeth of guinea pigs have a constant rate of growth in health. Deprivation of vitamin C causes the teeth to cease growing. Readministration of the vitamin restores the growth. Administration of small amounts of antiscorbutic substance results in rates of growth roughly proportional to dosage. Under standard experimental conditions used in the testing of food stuffs for antiscorbutic value, the rate of tooth growth would appear to be a precise indication of the degree of scurvy, being more delicate than the Sherman score and more constant as well as more simple than the Höjer method. Stress in terms of usage appears to exaggerate the scorbutic lesions in the teeth.

A Method of Detecting Sparsely Distributed Tubercle Bacilli. Joseph Felson. Arch. Path., x (1930), 1, p. 110.

The principle of this method for detecting sparsely distributed tubercle bacilli consists in increasing the specific gravity of a given specimen by means of sodium chlorid (1.5 to 2 per cent) allowing the preparation to stand, and skimming off the surface area in which the bacilli are concentrated, by means of a large right-angle loop. Experimentally it was found that a 4-mm loopful of positive tuberculous sputum, suspended in 250 cc of hypertonic saline solution, invariably gave more positive results than a suspension of the same material in distilled water. Rather high saline concentrations failed to alter materially the microscopic appearance of the tubercle bacilli.

Results Following Intrarenal Arterial Tuberculin Injections in Normal and Tuberculous Monkeys, Goats and Swine. Esmond R. Long, Charles B. Huggins and Arthur J. Vorwald. Amer. Jour. Path., vi (1930), p. 449.

Distinct renal allergic responses were secured on the injection of tuberculin protein into the renal arteries of tuberculous monkeys, goats and swine. The allergic nature of the response was established by the fact that similar injection into normal controls did not cause injury (except such as occurred from vascular occlusion). In the monkeys the lesion produced was purely degenerative, in the goats chiefly degenerative but occasionally inflammatory, and in the swine degenerative and of more inflammatory nature than in the goats. The inflammation in the goats and swine took the form of an interstitial nephritis. The glomerular changes observed in a former investigation, in which larger dosage of tuberculin protein was used, were not produced. The intensity of renal tuberculin reaction did not parallel the intensity of cutaneous reaction in this series of animals.

Tissue Reactions in Rabbits Following Intravenous Injection of Bacteria. Robert N. Nye and Frederic Parker, Jr. Amer. Jour. Path., vi (1930), 4, p. 381.

Following the intravenous injection into rabbits of relatively large doses of dead bacteria, there is a marked reaction of the tissues which contain cells of the reticulo-endothelial system. This reaction consists in an increase of lymphoid cells, which are eventually transformed into, or replaced by, monocytes and giant cells. Such lesions ordinarily are temporary and result in no permanent damage. Identical lesions occur after the intravenous injection into rabbits of various colloidal substances. Such changes represent the reaction of normal rabbits in the disposition of foreign materials in the blood-stream and have nothing to do with reactions secondary to sensitization or immunization.

Epizootic Fox Encephalitis. I. General description. R. G. Green, N. R. Zeigler, B. B. Green and E. T. Dewey. Amer. Jour. Hyg., xii (1930), 1, p. 109.

Epizootic fox encephalitis is a common disease of the silver or red fox in captivity. The infection is caused by a filtrable virus which invades the blood and may be demonstrated in the various tissues. Loss of appetite, convulsions, hyperexcitability, lethargy, paralysis, muscular twitchings, hallucinations, weakness and coma are some of the symptoms produced. The typical gross lesions of the disease are hemorrhages, which may be present in

one or more organs. Such hemorrhages are local extravasations of blood. The tissues in which gross hemorrhages are of the greatest significance are those of the central nervous system. Occurrences of gross hemorrhages in the central nervous system are to be considered occasional exaggerations of constantly occurring microscopic hemorrhages, which are a major characteristic of the disease. The general microscopic picture is one of hemorrhage and round-cell infiltration, but those changes appear to be more significant as a general reaction of nerve tissue to injury than as a reaction typical of this disease. Mortality in a natural epizootic usually runs from 12 to 20 per cent and is usually greater among young stock. The disease has been experimentally transmitted to foxes, mink and dogs, but carefully conducted experiments have failed to infect rabbits, white rats or ferrets. The authors also discuss the various phases of immunity.

Action of Formaldehyde on the Aggressive Substance of Blackleg Filtrate, Bacterin and Aggressin. Joseph P. Scott. Jour. Inf. Dis., xlvi (1930), 6, p. 460.

Concentrations of 0.5 per cent formaldehyde increased blackleg aggressin, filtrates and bacterins to their greatest potency. The increase in potency produced in these products is dependent on the amount of aggressive substance developed in the culture from which the product is made. Formaldehyde apparently acts on the aggressive substance in a quantitative manner. The presence or absence of bacterial cells in the product has no effect on the potency. It is shown that the amount of growth, the production of aggressive substances and the pathogenicity of a blackleg culture are independent. Virulent blackleg cultures consists of three parts: (1) living bacterial cells, (2) aggressive substances or products of growth produced during the development of cultures of *Clostridium chauvaei* and (3) a lethal substance or specific activity of the virulent bacterial cells. Potent blackleg products should have a potency of at least 20 aggressive units per dose. By the use of certain mineral salts and of the proper amount of formaldehyde, blackleg filtrates and bacterins having potencies of 130 and 260 aggressive units can be produced. Formaldehyde inhibits the agglutinogen but increases the activity of the protective antigenic complex.

ON THE PRESENCE OF THE NON-PATHOGENIC TRYPANOSOME MELOPHAGIUM IN THE BLOOD OF VICTORIAN SHEEP, AND ITS TRANSMISSION BY MELOPHAGUS OVINUS. A. W. Turner and D. Murnane. Aust. Jour. Exp. Biol. & Med. Sci., vii (1930), p. 9.

Trypanosoma melophagium is one of the larger of the mammalian trypanosomes and has been found in sheep in Victoria. Examinations of the gut of the sheep-ked, *Melophagus ovinus* L. have shown an enormous infection in the posterior three-quarters or two-thirds portion with the trypanosomes. Ked-free lambs, raised from birth in the laboratory and kept away from infected sheep, have remained free from keds and trypanosomes. Such lambs, fed an emulsion of ground-up abdomens of some hundred of infected keds, have shown trypanosomes in blood cultures from the sixth day onwards after feeding. Pre-infectional splenectomy appears to increase the number of circulating trypanosomes.

SELECTIVE MEDIA FOR THE CULTIVATION OF B. PULLORUM AND B. SANGUINARUM. W. R. Kerr. Jour. Comp. Path. & Therap., xliii (1930), 1, p. 77.

Brilliant green inhibits a great many of the coliform group which are normal saprophytes in the intestinal canal of even very young chicks, and it also inhibits all Gram-positive organisms. Brilliant-green media were used successfully for the diagnosis of carriers among adult fowls in cases where a chronic enteritis exists and yet the organism could not be found in the ovaries. *B. pullorum* was isolated by means of this selective medium from carcasses in all stages of putrefaction, up to and including thirty days after death. *B. pullorum* remained viable in moist feces for a period of 101 days. Brilliant green was used also in the diagnosis of fowl typhoid and in the demonstration of "carriers" of this disease.

EFFECT OF DYES ON THE VIRUS OF INFECTIOUS CHICKEN TUMOR No. 1. Margaret Reed Lewis. Amer. Jour. Hyg., xii (1930), p. 288.

The causative agent of the chicken tumor No. 1 may be inactivated in the tumor extract by a number of the common biological dyes. Some of these dyes brought about an unfavorable hydrogen-ion concentration in the solution containing the virus. Of those that did not produce an unfavorable hydrogen-ion

one or more organs. Such hemorrhages are local extravasations of blood. The tissues in which gross hemorrhages are of the greatest significance are those of the central nervous system. Occurrences of gross hemorrhages in the central nervous system are to be considered occasional exaggerations of constantly occurring microscopic hemorrhages, which are a major characteristic of the disease. The general microscopic picture is one of hemorrhage and round-cell infiltration, but those changes appear to be more significant as a general reaction of nerve tissue to injury than as a reaction typical of this disease. Mortality in a natural epizootic usually runs from 12 to 20 per cent and is usually greater among young stock. The disease has been experimentally transmitted to foxes, mink and dogs, but carefully conducted experiments have failed to infect rabbits, white rats or ferrets. The authors also discuss the various phases of immunity.

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The causative agent of the chicken tumor No. 1 may be inactivated in the tumor extract by a number of the common biological dyes. Some of these dyes brought about an unfavorable hydrogen-ion concentration in the solution containing the virus. Of those that did not produce an unfavorable hydrogen-ion

concentration, toluidin blue, eosin, erythrosin, phenol indophenol and dichlorophenol indo-phenol exhibited great inactivating power on the virus. Toluidin blue inactivated the virus of the chicken sarcoma in solutions containing about one part of dye to 10,000 parts of a highly virulent tumor extract.

THE INFECTION OF RABBITS WITH THE ANTHRAX BACILLUS BY WAY OF THE TRACHEA. Studies on the defensive and metabolic apparatus of the lungs. B. M. Fried. Arch. Path., x (1930), 2, p. 213.

The intratracheal introduction of anthrax bacilli causes no disease in rabbits provided the extrapulmonary tissues are spared contamination. The microörganisms so inoculated are retained by the lungs and are destroyed within a few hours after injection. The destruction of the highly pathogenic bacilli is performed by macrophages by way of phagocytosis. The cells found in groups alongside the wall of the air-sacs (so-called alveolar epithelium) play the outstanding rôle in the phagocytosis of the anthrax bacilli. The experiments have shown likewise that the pulmonary reaction to this "virtual" infection is akin to the "model infections" with vital dyes and oils reported in earlier studies.

THE EFFECT OF RACHITIC DIETS ON EXPERIMENTAL TUBERCULOSIS. Agnes H. Grant. Abst. Arch. Path., x (1930), 2, p. 296.

Resistance to tuberculosis can be decreased from a natural immunity to a moderate susceptibility, without destroying the growth impulse, by prolonged disturbance in the optimal balance that should exist between the calcium, vitamin C and vitamin D of the diet. A prolonged excess of vitamin D in the diet, either with a deficiency of calcium or with a normal amount, hastens the spread of tuberculosis through the lungs. The substitution of vitamin C for vitamin D at the time of inoculation, or the addition of vitamin C to a diet which is deficient in vitamin D, tends to increase the amount of tuberculosis in the lungs, rather than decrease it. The addition of vitamin C to a diet containing vitamin D tends to correct the diet, and to increase the resistance to tuberculosis. The degree to which diets corrected after inoculation can retard the spread of tuberculosis in white rats depends on the type of deficiencies and excesses in the diet before inoculation, and on how long the deficient diets have been given.



ARMY VETERINARY SERVICE

Regular Army

The following officers of the Veterinary Corps were transferred to the retired list, as of September 30, 1930:

Colonel Ray J. Stanclift, upon completion of 30 years' service.
Major Ralph M. Buffington, for physical disability.

Veterinary Reserve Corps

New Acceptances

Crawford, Andy William...2nd Lt....Vet. Hospital, Kansas State Agricultural College, Manhattan, Kansas.
Gatbonton, Alfredo Reyes..2nd Lt....Canaba, Pampanga, P. I.

Promotions

Adams, John Dawson.....	to Capt....	909 N. 15th St., Boise, Idaho.
Campbell, Ora Lee.....	Lt. Col.	Astoria, Ill.
Lane, James Aubrey (C)....	1st Lt...	Box 13, Wilberforce, Ohio.
Maus, Frank Charles.....	Capt....	52 Park Place, Johnson City, N. Y.
Moe, Lewis H.....	1st Lt...	225 Lowry St., Stillwater, Okla.
McLeod, Wm. Max.....	Capt....	1114 Bertrand St., Manhattan, Ks.
Nelson, Marvin Lothard....	Capt....	R. F. D. No. 1, Arkdale, Wis.
Rife, Charles Cornelius.....	1st Lt...	420 Edgewood Ave., N. E., Atlanta, Ga.

Southern Business Men Favor Tick Eradication

Business men, as well as the producers of live stock, are interested in cattle-tick eradication in many of the southern states, according to reports received by the U. S. Department of Agriculture. In Florida, one of the leading banking institutions recently used paid advertising space in a daily newspaper, to comment favorably on the progress being made in tick eradication as follows:

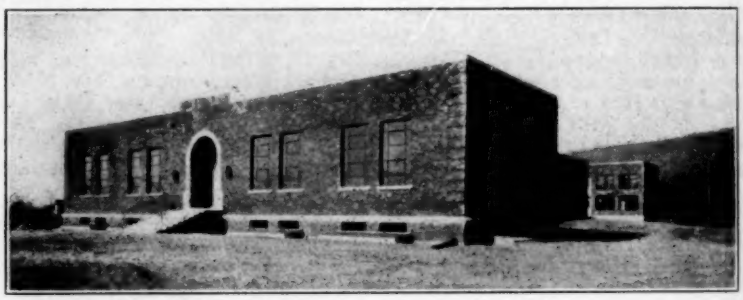
> Many of the leading dairy and cheese interests of the North and West have been establishing plants throughout the South, thus helping to put the dairy industry on a safe and solid foundation, and to encourage the diversification of agriculture, the raising of more and better live stock, and the production of milk, cream, butter and cheese. The tick can be abolished, and when this is accomplished Florida will quickly attain high rank as a producer of beef cattle and dairy products—and a new stream of dollars will flow into the State.



Pennsylvania Bureau of Animal Industry Laboratory Increases Its Staff

Dr. H. M. Martin (U. P. '16) has recently become associated with the Pennsylvania Bureau of Animal Industry as parasitologist. Dr. Martin's experience and training especially qualify him for this branch of the Pennsylvania Bureau of Animal Industry's activities in the investigation and control of animal diseases, including the diseases of poultry and game-protected animals and birds.

Following his graduation from the University of Pennsylvania, Dr. Martin spent a year in his Alma Mater in graduate study in



Main laboratory building, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.

pathology and bacteriology, and at the same time assisted one of Philadelphia's practitioners in general veterinary practice. After the completion of his graduate work at Pennsylvania, he was appointed instructor in veterinary science in the University of Wyoming. After spending a year at this institution, he resigned to accept a position as assistant professor of animal pathology and hygiene at the University of Nebraska and was later promoted to an associate professorship.

During his stay in Nebraska he pursued graduate work in zoology and other subjects. In 1923 he received the degree A. M. and in 1927 the degree Ph. D. was granted. While at the Uni-

versity of Nebraska, Dr. Martin's work consisted almost entirely of research in animal pathology and allied subjects. His work on tuberculosis and parasites is outstanding. His major work in zoology consisted of parasitology.

Dr. Martin was associated with Dr. L. Van Es while in Nebraska, and collaborated with him in the preparation of numerous publications in the field pathology, bacteriology and parasitology. Pennsylvania Bureau officials consider that their organization was especially fortunate in securing Dr. Martin.

Other additions to the laboratory staff at Harrisburg include Dr. W. E. Martindale (U. P. '28), who spent one year as Resident Surgeon in the University of Pennsylvania Veterinary Hospital; Dr. R. H. Alexander (K. S. A. C. '30), Dr. P. H. Seitz (U. P. '30), Dr. G. T. Price (Ont. '30) and Dr. J. B. Stauffer (U. P. '30).

International Union Against Tuberculosis

The seventh conference of the International Union against Tuberculosis was held at Olso, Norway, August 13, 14 and 15, 1930. It was held under the patronage of the King and Queen of Norway. Three subjects were on the agenda of this conference: "Preventive Vaccination against Tuberculosis," "Thoracoplasty in the Treatment of Pulmonary Tuberculosis" and "The Teaching of Tuberculosis to Students and Doctors." One day was occupied with each subject. The chief reporter on the first subject was Dr. Calmette and the entire day was devoted to BCG. There were several co-reporters from various countries on the use of BCG. The afternoon was spent in a discussion of this subject. Dr. Calmette presented a résumé of the work done at the Pasteur Institute with BCG. He stated that anti-tuberculous immunity depended on the presence of a few specific bacilli that were not dangerous to the health of the host. He claimed that these conditions were fulfilled in BCG. Many others cited experiences in support of such a method. Dr. W. H. Park, of New York City, stated that they had dissociated BCG into two types of cultures and raised the question whether such cultures might not become virulent in animal bodies. Dr. E. A. Watson, of Ottawa, Canada, the only veterinarian appearing on the program, cited experiments showing that cultures of BCG has become virulent in guinea pigs and when grown in the presence of rabbit serum has become virulent for rabbits.

In the discussion of "The Teaching of Tuberculosis to Students and Doctors," it was brought out that no institution has a

separate chair for the teaching of tuberculosis but that many institutions were providing special facilities for the teaching of this disease.

American veterinarians attending this conference, which followed the 11th International Veterinary Congress, were: Dr. George Hilton, Veterinary Director-General, of Ottawa, Canada; Dr. E. A. Watson, of the Animal Diseases Research Institute, Ottawa, Canada, and Dr. E. L. Stubbs, Professor of Veterinary Pathology, University of Pennsylvania, Philadelphia, Pa. Seven hundred and fifty were registered for this conference.

E. L. S.

Tuberculosis Conference

A conference of regulatory officials and others interested in the control of tuberculosis of live stock and representing the states of Illinois, Indiana, Kansas, Michigan, Nebraska, New York, Ohio, West Virginia and Wisconsin, convened in the office of the Commissioner of Agriculture, Lansing, Mich., September 19, 1930.

Present: Dr. A. E. Wight, Chief, Tuberculosis Eradication Division, Bureau of Animal Industry, Washington, D. C.; H. R. Smith, Commissioner, National Live Stock Exchange, Chicago, Ill.; D. W. Robinson, Director, Bureau of Animal Industry, Springfield, Ill.; Dr. J. E. Gibson, Inspector-in-Charge, U. S. Bureau of Animal Industry, Indianapolis, Ind.; R. L. Cuff, Live Stock Commissioner, Kansas City, Kans.; H. E. Powell, Commissioner of Agriculture, Lansing, Mich.; J. E. Burnett, Director, Bureau of Animal Industry, Lansing, Mich.; Dr. T. S. Rich, Inspector-in-Charge, U. S. Bureau of Animal Industry, Lansing, Mich.; Dr. C. H. Clark, State Veterinarian, Lansing, Mich.; Dr. B. J. Killham, Extension Veterinarian, East Lansing, Mich.; Dr. H. Preston Hoskins, Secretary, American Veterinary Medical Association, Detroit, Mich.; Dr. W. T. Spencer, Live Stock Commissioner, Omaha, Nebr.; Dr. E. T. Faulder, Director, Bureau of Animal Industry, Albany, N. Y.; Dr. A. J. DeFosset, Inspector-in-Charge, U. S. Bureau of Animal Industry, Columbus, Ohio; Dr. C. McCandless, State Veterinarian, Columbus, Ohio; Dr. H. M. Newton, Inspector-in-Charge, U. S. Bureau of Animal Industry, Charleston, W. Va.; and Dr. W. Wisnicky, Director, Live Stock Sanitation, Madison, Wis.

Commissioner H. E. Powell and Dr. B. J. Killham were elected as Chairman and Secretary, respectively.

After a discussion which was participated in by representatives of each state, the following resolution was unanimously adopted.

It is recommended that as soon as deemed advisable by the state officials in charge, and in no instance later than the date upon which an entire state is declared a Modified Accredited Area, regulations or legislation be made effective to require that all cattle entering the state affected, or any of the counties or townships thereof, except cattle for immediate slaughter, be properly and satisfactorily identified as having originated in Modified Accredited Areas, or be subject to an approved tuberculin test prior to such entrance.

B. J. KILLHAM, *Secretary.*

Michigan Achievement Celebration

A meeting that is believed to be unique in the history of animal disease control in the United States was held at East Lansing, Mich., on September 19. On that day Michigan celebrated a victory over an animal disease, namely, bovine tuberculosis. Those who gathered to celebrate the victory included live stock sanitary control officials of the federal government and eleven states, practicing veterinarians, breeders, dairymen, educators, county agents, health officers, members of the Michigan State Farm Bureau, members of the State Grange, and others interested in the live stock industry. The members of the Hillsdale County Board of Supervisors attended in a body.

The event culminated in a banquet held in the Union Building, Michigan State College, in the evening, with Professor Howard R. Smith, Commissioner, National Live Stock Exchange, Chicago, acting as toastmaster. Professor Smith directed attention to the fact that Michigan was the third state to complete the successful tuberculin-testing of all cattle, North Carolina and Maine having been the first and second, respectively. It was pointed out that Michigan was really the first prominent dairy state to reach the coveted goal. This was accomplished in a little over nine years, and represented the tuberculin-testing of more than 1,500,000 head of cattle.

Addresses were made by Hon. H. E. Powell, Commissioner of Agriculture, Lansing; Mr. M. L. Noon, President, Michigan State Farm Bureau, Jackson; Mr. C. L. Hill, Chairman, Department of Agriculture and Markets, and President, National Dairy Show, Madison, Wisconsin; Mr. H. R. Davison, Vice-President, Institute of American Meat Packers, Chicago, Illinois, and Dr. D. C. Lochead, Health Officer, Rochester, Minnesota.

Nebraska Meeting

Dr. B. Witt, secretary of the Nebraska State Veterinary Medical Association, has announced an interesting program for the annual meeting to be held at Lincoln, December 9-10. Among those who will contribute to the program are Dr. E. A. Benbrook, Iowa State College, Ames, Iowa; Dr. C. H. Hays, State Veterinarian, Lincoln; Dr. A. F. Schalk, Ohio State University, Columbus, Ohio; Dr. A. T. Kinsley, Kansas City, Mo.; Dr. B. A. Beach, University of Wisconsin, Madison, Wis.; Dr. C. P. Fitch, University of Minnesota, Saint Paul, Minn., and Dr. L. Van Es, University of Nebraska, Lincoln.

COMMUNICATION

PROMPT REPORT APPRECIATED

To the Editor:

I want to congratulate you on the publication of the proceedings of the sixty-seventh annual meeting of the A. V. M. A.

This is the most satisfactory report of an A. V. M. A. convention that has ever been published and I want to commend you for bringing out this splendid and inspiring report at such an early date.

A. D. Knowles.

Kelsey City, Fla., October 21, 1930.

Fiftieth Anniversary Commemorated

Dr. J. T. Glennon, of Newark, N. J., Second Vice-President, represented the American Veterinary Medical Association at the convocation to commemorate the fiftieth anniversary of the founding of the New Jersey Agricultural Experiment Station, New Brunswick, N. J., October 9, 1930.

On the same occasion Dr. J. Raymond Beach represented the University of California, and Dr. F. R. Beaudette the Kansas State Agricultural College.

A Cosmopolitan Faculty

Dr. C. H. Stange, Dean of the Division of Veterinary Medicine, Iowa State College, has always believed that a veterinary faculty should consist of members representing as wide a variety of schools as possible. Since the Division has been under his direction, there have been on the faculty graduates of most of the veterinary colleges of the United States. At present ten veterinary colleges and six other colleges and universities are represented. The veterinary colleges are: University of Pennsylvania, Ohio State University, Iowa State College, Alabama Polytechnic Institute, Colorado Agricultural College, Michigan State College, State College of Washington, and the universities of Buenos Aires, Munich and Zurich. The colleges or universities represented by graduates in science or other courses include Clemson College, Drake University, Cornell University, Des Moines University, University of Minnesota and the University of Leipsic.

AMERICAN VETERINARY MEDICAL ASSOCIATION

Proceedings of the Sixty-seventh Annual Meeting, Los Angeles, California, August 26 to 29, 1930

Section on General Practice

WEDNESDAY MORNING, August 27, 1930

The first session of the Section on General Practice was called to order at 9:30 a. m., by Dr. J. F. Rankin, chairman. (Dr. H. E. Kingman, secretary.)

CHAIRMAN RANKIN: I am very glad to be here, and I would like to take this opportunity to thank the gentlemen who have agreed to appear on this program. No one knows how hard it has been to arrange it, except Dr. Kingman and I. Dr. Kingman wrote to practically every man in the United States, excepting a few of the fellows in Oregon, Washington and British Columbia, and I tried to cover the rest. I didn't have any gray hairs until then; I have a few now. And you can see for yourself that Dr. Kingman is practically gray-haired. Both of us had to appear on the program to fill it up, and if our papers seem to bore you, gentlemen, you will know the reason.

I have nothing more to say, only that I am like the young gentleman who went out to make a speech. He took his mother along with him to hear his speech. When he had finished they started home. She didn't say anything, and he didn't say anything for quite a while. Finally he said, "Mother, how did you like my talk?" She said, "Well, son, there were only two things you forgot to do, and that was to shut up and sit down." (Laughter) So I will start the program immediately.

The following program was then presented:

"Sterility in Cows Caused by Endometritis," Dr. John F. Rankin.

"Endo-Cervicitis in the Cow," Dr. H. E. Kingman.

"The Part the Practitioner Plays in the Program for the Eradication of Bang Abortion Disease," Dr. W. H. Hendricks.

. . . The meeting recessed at 11:55 a. m. . . .

RECESS

THURSDAY MORNING, August 28, 1930

The second session was called to order at 9:25 a. m., by Chairman Rankin.

The following program was presented:

"Bovine Mastitis," Dr. Maceo E. Spratlin.

"Parturient Paresis," Dr. J. E. McCoy.

"Traumatic Reticulitis," Dr. Chas. H. Seagraves (Read by title.)

. . . The meeting adjourned at 11:30 a. m. . . .

ADJOURNMENT

Section on Sanitary Science and Food Hygiene

WEDNESDAY MORNING, August 27, 1930

The first session of the Section on Sanitary Science and Food Hygiene was called to order at 9:30 a. m., by Dr. W. J. Butler, chairman. (Dr. W. H. Lytle, Secretary.)

CHAIRMAN BUTLER: As you all know, the Chairman is supposed to make quite a speech. However, the other day I read that there were more chairmen and toastmasters in the United States who attended banquets and different meetings and who spoiled those meetings than anything else. I am not going to spoil this meeting by taking the time of the gentlemen who are going to read papers.

To any of the men in regulatory work this Section means a great deal. We have a great deal to do, and from my observation of all the work in regulatory affairs, the principal thing that we find out is to do our work properly and to do it with credit to the veterinary profession.

I heard a little story, not long ago, about hard times. You know when things are not going very well we have a tendency to howl. There was a banker from New York who wanted to find out about conditions throughout the country. He had heard they were rather bad, so we went to a little country town just to see how things were coming along.

He walked down the main street and then into the side streets. He was coming across a little lot, when he saw a dog sitting there, just howling. The banker said, "What's the matter, dog?" The dog didn't pay any attention but continued to howl. He howled and howled and howled.

Just then an old-timer happened to be passing by and the banker said, "What's the matter with that dog?"

He said, "I don't know just what is the matter."

The banker said, "What do you think is the matter with him?"

"He is just sitting on a burr."

The banker said, "Why doesn't he get off the burr?"

"It's a whole lot easier to sit on the burr and howl than it is to get off and do something." (Laughter)

That's the way it is with us. Sometimes it is easier to find fault than it is to go ahead with what we have to work with.

There is one thing I do want to mention to you now, and that is, in all of this work we want to stay down close to earth. A great many of our states are getting out regulations that are going to be hard to follow, and before we get out a regulation restricting the movement of live stock, we want to know just where we are and what that regulation is going to accomplish, and whether or not it is practical and can be enforced.

I think one of the fundamental things before the live stock sanitary officials today is to know that the regulations are sound and that they can be enforced, and also that they are practical.

We have talked of uniform regulations for the last twenty years. I am of the opinion that it is pretty nearly impossible for all the states to have uniform regulations. Different states require different regulations. Geographic conditions govern, to a certain extent, the kind of regulations that you must get out, but we do want to get basic regulations that are sound, and we don't want to fly off before we know just what we are doing.

The following program was then presented:

"Some Notes on Liver Fluke Investigations," Dr. J. N. Shaw.

"A Review of Tuberculosis Control in California," Dr. J. P. Iverson.

"Control of Anthrax in Live Stock of the Northwest," Dr. E. R. Derflinger.

"Actinobacillosis," Dr. A. J. Damman.

"Tularemia," Dr. R. R. Parker. (Read by title.)

. . . The meeting recessed at 12:25 p. m. . . .

RECESS

THURSDAY MORNING, August 28, 1930

The second session was called to order at 9:15 a. m., by Chairman Butler.

The following program was presented:

"The Detection of Horse Meat as an Adulterant in Sausage, and Other Studies of the Precipitation Test." Dr. F. M. Bolin.

"Recent Progress in Duck Disease Studies," Mr. Paul A. Shaw.

"Sanitary Science, the Basis for Abortion Control," Drs. Walter Wisnicky and V. S. Larson. (Read by Dr. Larson.)

"The Practical Side of Meat Inspection," Dr. J. G. Jervis.

"Brucelliasis in Swine at the University of California with an Epidemiological Report of the Infection of Two Attendants," Drs. J. A. Howarth and F. M. Hayes. (Read by Dr. Howarth.)

"Epidemiologic Data of Brucelliasis—Report of Two Human Cases," Dr. J. C. Geiger.

. . . The meeting adjourned at 12 noon. . . .

ADJOURNMENT

Section on Education and Research

WEDNESDAY MORNING, August 27, 1930

The first session of the Section on Education and Research was called to order at 9:30 a. m., by Dr. B. T. Simms, of Corvallis, Oregon, who had been appointed Chairman pro tem, by President Ferguson, in the absence of Dr. H. E. Biester. (Dr. Jacob Traum, of Berkeley, Calif., was appointed Secretary pro tem., by President Ferguson, in the absence of Dr. W. A. Hagan.)

The following program was presented:

"The University of California Committee for the Study of Brucella Infections," Dr. C. M. Haring.

"Fertility Studies in the Bull," Drs. C. R. Donham and B. T. Simms. (Read by Dr. Simms.)

"The Incidence of *B. Abortus* Agglutinins in Human Sera," Dr. R. A. Whiting.

"Coccidioidal Granuloma in Animals," Dr. Jacob Traum and Dorothy Beck. (Read by Miss Beck.)

"Coccidioidin as a Diagnostic Agent in Man and Animals," Dorothy Beck and Dr. Jacob Traum. (Read by Miss Beck.)

"Correlation of Blood Reactions with the Breeding Records over a Period of Years in an Abortion-Infected Herd of Cattle," Dr. C. F. Clark. (Read by Dr. E. T. Hallman.)

. . . The meeting recessed at 12:25 p. m. . . .

RECESS

THURSDAY MORNING, August 28, 1930

The second session was called to order at 9:25 a. m., by Acting Chairman Simms.

The following program was presented:

"The Museum of Pathology: A Neglected Institution," Dr. William H. Feldman. (Read by Dr. H. C. H. Kernkamp.)

"*Salmonella Enteritidis* Infection in Guinea Pigs and Rabbits," Drs. R. C. Duthie and C. A. Mitchell. (Read by Dr. E. A. Bruce.)

"Histopathology of the Central Nervous System in Hog Cholera," Dr. Oskar Seifried.

"The Bacillus of Swine Erysipelas Associated with Arthritis in Lambs," Dr. Hadleigh Marsh. (Read by Dr. Jacob Traum.)

"Chloroform-Treated Rabies Vaccine," Major R. A. Kelser. (Read by Dr. Maurice C. Hall.)

"Histological Studies on A-Avitaminosis and Coryza in Chickens," Dr. Oskar Seifried. (Same paper as presented before Section on Poultry.)

"Salmon Poisoning," Drs. B. T. Simms, C. R. Donham, J. N. Shaw and A. M. McCapes. (Read by title.)

. . . The meeting adjourned at 11:45 a. m.

ADJOURNMENT

Section on Small Animals

WEDNESDAY MORNING, August 27, 1930

The first session of the Section on Small Animals was called to order at 9:30 a. m., by Dr. C. W. Bower, chairman.

CHAIRMAN BOWER: This, I believe, is the fifth meeting at which the Section on Small Animals has met officially. We had, at St. Louis, a Section on Small Animals which was non-official; it was in connection with the Section on General Practice. Our first official meeting was at Portland, some five years ago. This Section has been increasing and growing in popularity all the time.

I am rather disappointed this year that there are so many vacant seats. When I first looked into the room I thought that we had entirely too small a room, and I still think that we have. I believe, before the session is over, we will have to get into a bigger assembly room, because generally this is the most popular section and the largest.

It is unfortunate, indeed, that our Secretary, Dr. Schlotthauer, of the Mayo Foundation, has not been able to attend this session. He says that due to the geographical location of the meeting, and to the increase in work right at this particular time, he was unable to get away.

I want to say this to the credit of Dr. Schlotthauer—that he has done a tremendous amount of work in preparing this program. As you know, the Secretary gets all the credit for preparing the program but he is the fellow who really does the work. He has had a hard time this year, particularly because of the location of the meeting and the fact that so many veterinarians are taking the European trip. Time and again he wrote me and sent me tentative programs and then time and again he would have to write and change these programs, because somebody would promise him a paper and then find that he would not be able to appear.

The first thing on the program today is the Chairman's address. There will be none. That is absolutely non-essential, I think, in a section that has been established as long as this one has.

The next is the Secretary's report. I will read it.

. . . Chairman Bower read the report, which was ordered filed. . . .

Report of Secretary

MR. CHAIRMAN AND MEMBERS:

Because of the extreme western location of the 1930 meeting place for the sixty-seventh annual meeting of the A. V. M. A., general business depression throughout the country, and the fact that the International Veterinary Congress met in Europe this summer, many men now actively engaged is small-animal practice can not be here to present papers or discuss those of their colleagues. However, we fortunately have an excellent program. As you will note, the program deals with nearly all of the important branches of small-animal practice. It should prove interesting to all.

Judging from the literature and personal visits with men engaged in small-animal practice in various parts of the country, it appears to me that the men thus engaged are doing an excellent work. Through their efforts small-animal practice has been elevated to a position of stupendous importance. People everywhere are rapidly learning that the trained veterinarian possesses the exact knowledge required to enable them to keep their pets and small animal servants in a state of health, usefulness and happiness. They are very grateful for this. The kindly druggist and the "practical" kennel men are no longer doctoring the dogs of the world. This is as it should be. However, let us not be satisfied. We must all carry on. This can best be done by helping each other. Tell your fellow practitioner of any new and better method of prophylaxis or treatment of a disease that you may discover. It is only by such cooperative efforts that we can all continue to grow.

(Signed) C. F. SCHLOTTHAUER.

The following program was presented:

"Recent Advances in the Prophylaxis and Treatment of Canine Distemper," Dr. F. W. Wood.

"Clinical Manifestations of Pathological Conditions of the Genital Organs of Small Animals," Dr. James B. Jones.

"Small-Animal Hospitalization," Dr. W. E. Frink.

. . . The meeting recessed at 12:30 p. m. . . .

RECESS

THURSDAY MORNING, August 28, 1930

The second session was called to order at 9:30 a. m., by Chairman Bower.

The following program was presented:

"Canine Obstetrics," Dr. H. A. Gould.

"Diseases of the Mouth," Dr. E. A. Ehmer.

* * * *

CHAIRMAN BOWER: I have here a communication from Dr. J. V. Lacroix, of Evanston, Ill., that I would like to read, to see what action you desire to take on it.

. . . Chairman Bower read the communication, which was on the subject of the muzzling of dogs. . . .

CHAIRMAN BOWER: You have heard the communication. What is your pleasure?

DR. J. C. FLYNN: I move that it be the sense of the Section on Small Animals that we adopt the resolution presented by Dr. Lacroix and convey this information to the Association at the next general session for approval. In other words, I would like to see this Section adopt the resolution, subject to the approval of the entire Association.

CHAIRMAN BOWER: I do not know whether this is a resolution or not. It seems to be merely the consensus of opinions of several veterinarians, that muzzling is not practical.

DR. FLYNN: Let us go on record as being opposed to the muzzling of dogs, for the protection of the public against rabies.

CHAIRMAN BOWER: Would you have this referred to a general session?

DR. FLYNN: I think it should be.

CHAIRMAN BOWER: At this meeting?

DR. FLYNN: Yes. It would be out of place for our Section to adopt it and then have it go no further.

CHAIRMAN BOWER: Do I hear a second to the motion?

DR. O. J. KRON: I second the motion.

DR. J. F. MCKENNA: I think a word or two should be added. I believe that the motion was that we go on record as being opposed to the muzzling of dogs to protect people against rabies. I would suggest that it read that we are opposed to the muzzling

of dogs as being a means of protecting people against rabies. There is a lot of difference in the wording of the two.

DR. FLYNN: We certainly do not want it to appear that we are opposed to preventing rabies.

CHAIRMAN BOWER: I think that is a good suggestion. Any further discussion?

. . . The motion was put to a vote and carried. . . .

* * * *

The program was then resumed and the following papers presented:

"Bulla-Osteotomy in the Dog," Drs. G. W. McNutt and J. E. McCoy. (Presented by Dr. McNutt.)

"Some of the Uses of Normal Saline Solution Infusion in Canine Practice," Dr. H. Calvin Rea. (Read by title.)

"Care and Feeding of Small Mammals in a Zoological Park," Dr. C. V. Noback. (Read by title.)

. . . The meeting adjourned at 12:05 p. m. . . .

ADJOURNMENT

Section on Poultry

WEDNESDAY MORNING, August 27, 1930

The first session of the Section on Poultry was called to order at 9:30 a. m., by Dr. W. R. Hinshaw, chairman.

CHAIRMAN HINSHAW: The first thing on the program is the Chairman's address. As the Chairman has no address, we will proceed with the program. In the absence of Dr. Biltz, of Pennsylvania, secretary of the Section, President Ferguson has appointed Dr. W. T. Johnson, of Corvallis, Oregon, to act as Secretary pro tem.

The following program was presented:

"Accuracy of Three Cooperating Laboratories in Detecting Pullorum Disease by Agglutination Test," Dr. J. Biely. (Read by Dr. E. M. Dickinson.)

"Some Recent Studies in Poultry Diseases," Dr. George Kernohan.

* * * *

CHAIRMAN HINSHAW: Secretary Hoskins and I have discussed the desirability of presenting the report of the Special Committee on Poultry Diseases at this time. It was to have been presented at the general session this afternoon. This morning it was decided to present the report in full to the Section

on Poultry, then get a vote on it by this group, and have it read by title at the next general session.

. . . Dr. Hinshaw read the report*. . . .

CHAIRMAN HINSHAW: A copy of this report has been sent to each member of the Committee, and all have approved it except Dr. Beach. He has not returned his copy, so I take it for granted that he approves the report.

SECRETARY JOHNSON: I move that the report be received and referred to the general session.

. . . The motion was seconded, put to a vote and carried.

. . .

* * * *

. . . The meeting recessed at 10:30 a. m.

RECESS

THURSDAY MORNING, August 28, 1930

The second session was called to order at 9:10 a. m., by Chairman Hinshaw.

The following program was presented:

"Studies in the Pathology of Avian Coccidiosis," Dr. H. J. Stafseth.

"Histological Studies on A-Avitaminosis and Coryza in Chickens," Dr. Oskar Seifried.

"Coccidiosis of the Chicken with Special Reference to Species," Dr. W. T. Johnson.

"A Note on Avian Laryngotracheitis," Drs. Robert Graham, Frank Thorp, Jr. and W. A. James. (Read by Dr. W. H. Welch.)

"The Use of Nicotin and Its Compounds for the Control of Poultry Parasites," Dr. C. D. Carpenter.

"*Clostridium Welchii* Infection in the Domesticated Fowl," Dr. K. W. Niemann.

. . . The meeting adjourned at 11:50 a. m. . . .

ADJOURNMENT

Erratum

The correct time for the broadcasts of Dr. H. W. Brown, over Station WOWO, under the auspices of the Battle Creek Dog Food Company, is 7:45 p. m. C. S. T., instead of 7:20, as announced in the October issue of the JOURNAL.

*Published in the JOURNAL, October, 1930, pp. 503-506.

Report of the A. V. M. A. Committee Appointed to Confer with the Surgeon General, U. S. Public Health Service

The committee appointed by President Ferguson to confer with Surgeon General Hugh S. Cummings, U. S. Public Health Service, and representatives of medical, dental, pharmaceutical, veterinary and other scientific associations and agencies, met at Washington, D. C., Tuesday, August 12, 1930.

The convention was called to consider the question of (a) the necessity for, and (b) the methods to be used in carrying out the provisions of the act approved June 14, 1930, relating to the quantities of crude opium, coca leaves, and their salts, derivatives and preparations, together with such reserves thereof as are necessary to supply the normal and emergency medicinal and scientific requirements of the United States.

The impression your committee had from the conference was that the conference was called for the purpose of curbing the use of opiates and cocain narcotics, and to consider the legitimate narcotic needs for the United States.

The objects of the conference were, furthermore, to determine the necessary amounts of coca leaves and crude opiates to be imported for the use of the professions, plus a reasonable amount for emergency without accumulating a dangerous reserve. There were no indications of putting any limits on the legitimate use of drugs.

The following points were developed:

1. A complete analysis of hospital records with a request for the American Medical Association's cooperation to study normal medicinal and scientific requirements.
2. A detailed examination of prescriptions to determine the ill-advised use of narcotic drugs.
3. An analysis of records of dispensing physicians.
4. An analysis of the use and sale of the so-called exempt preparations.
5. A field study of morbidity and mortality with a view to forecasting the extent and character of sickness to be expected.
6. An educational program with endeavors to reduce the demand for narcotic drugs, and substituting the non-narcotic drugs for them.

The whole spirit of the conference seemed to be one of cooperation with no attempt to tell anybody how much of these drugs should be used; neither was the subject of enforcing the law brought up.

(Signed) J. P. TURNER, *Chairman*
(Signed) H. J. MILKS
(Signed) R. S. AMADON



MICHIGAN DIVISION, NATIONAL ASSOCIATION OF B. A. I. VETERINARIANS

At a special meeting of the Michigan Division of the National Association of B. A. I. Veterinarians, held in Detroit, July 1, 1930, Dr. W. L. Chandler, a prominent figure in scientific circles, and, at present, Professor of Parasitology at the Michigan State College, discussed quite interestingly the product known as colloidal iodin, which he developed. The meeting was attended by a number of the veterinarians on the meat inspection staff of the Detroit Department of Health.

On August 5, 1930, Dr. L. B. Sholl, also of the Michigan State College, very instructively presented the subject of "Mastitis" to the Association.

Spirited discussions followed, at both meetings.

J. E. ZELZTER, *Secretary.*

MARYLAND STATE VETERINARY MEDICAL ASSOCIATION

The semiannual meeting of the Maryland State Veterinary Medical Association was held at the University of Maryland, College Park, July 24-25, 1930, with about seventy in attendance.

The meeting was called to order by our genial President, Dr. Hulbert Young, of Baltimore. The presidential address was short and to the point. It consisted of a review of the important developments in the veterinary field during the past few months. Attention was called to the efficiency of the work being carried on in the State, as shown by the healthy condition of the animal population. Some of the problems facing the profession were pointed out. The chief one, from the legislative standpoint, is the proposal by the agricultural interests, that the accredited herds be tested by the State. This is not in conformity with the understanding entered into by the practitioners when the work was started. It is hoped that an amicable solution may be found.

The second paper was entitled, "The Control of Bovine Infectious Abortion or Bang Disease." It was delivered by Dr. C. P. Fitch, University Farm, Saint Paul, Minnesota. Dr. Fitch was at his best, and I am sorry that the paper cannot be reported in full. Among other things, Dr. Fitch said:

> The control of any infectious disease, whether of man or animals, depends fundamentally upon the biology of its cause. Also, to a considerable extent, upon the educational methods that are used to bring to the people, the information necessary to carry out effective control measures.
>
> Although there have been many causes of abortion in cattle described, the Bang organism is the most important, and in our opinion is responsible for approximately 85 per cent of the total abortions occurring among bovines. The control of the disease cannot be brought about through the application of any medicines, nor can the disease be cured with now known therapeutic agents. Vaccines, bacterins and other biological agents have been used extensively for the control of this infection, due to this germ. The results show that bacterins produce very little resistance to the disease. Living vaccines do increase the resistance of animals to the infection, but individuals vary markedly in their ability to produce such resistance following the injection of these products. The danger of the transmission of the germ to the human family, through the milk of infected animals, renders the use of such products unsafe.
>
> We believe that the only method that has been demonstrated, up to the present time, as effectively controlling the disease, is the clean herd on the basis of the blood test.

The third paper was entitled, "Contagious Abortion Work in a Large Dairy Herd," by Dr. J. W. Hughes, of Ammendale, who stated that the paper did not purport to be a technical discussion of the pathology of Bang disease, but was rather a compilation of a part of the data obtained from the management and treatment of an infected herd, and certain conclusions based thereon.

The fourth paper was entitled, "The Effect of Age and Temperature upon the Agglutinability of Bovine Sera," by Dr. L. J. Poelma, of the University of Maryland. The paper will appear in an early number of the *Cornell Veterinarian*. It is, therefore, not reported here.

The last paper of the morning program was entitled, "The Channels of Infection of *Brucella Abortus* Bang in Guinea Pigs," by Dr. C. L. Everson, of the University of Maryland. It will be published later.

The afternoon was devoted to clinics. Dr. F. B. Dibbell, of Baltimore, conducted the small-animal work. Dr. J. N. Frost, of Cornell University, had charge of the large-animal part. Both branches were most interesting and instructive.

The banquet was held in the evening, at the University Dining Hall. Dr. Hulbert Young, President, acted as toastmaster. The speakers outside of the profession were Hon. S. M. Shoemaker, chairman of the Board of Regents, University of Maryland; Dr.

R. A. Pearson, president of the University; and Mr. I. W. Heaps, secretary and general manager of the Maryland State Dairymen's Association. There were about sixty-five in attendance.

The first paper on the second day was given by Dr. J. J. Jones, LaPlata. His topic was, "Better Cooperation." The second paper was delivered by Dr. E. S. Savage, Professor of Animal Husbandry, Cornell University. It was entitled, "The Need of Dairy Cows for Protein." This paper was based on the cooperative experiments being conducted at Cornell and will be published later.

The third paper was entitled, "Diseases of the Joints and Tendon Sheaths," by Dr. J. N. Frost, of Cornell University. This paper also will be published later. The fourth paper was entitled, "Field Trials and Field Trial Dogs," by Mr. Ford Young, of Washington, D. C. Mr. Young's paper will also be published later.

The last paper of the session was entitled, "The Testing of Accredited Herds," by Dr. E. B. Simonds, in charge of tuberculosis eradication, Baltimore. Dr. Simonds discussed one of the most perplexing problems facing the Association. The demand of the dairymen that the State test the accredited herds without charge, will prove a serious blow to the practitioners, if it should become law and no way is found to turn the work back to the practitioners. This, of course, will react adversely on the dairy industry.

A general discussion of the subject followed Dr. Simond's paper. Committees were appointed and instructed to work out ways and means of handling the situation.

The last afternoon was devoted to large-animal clinics. Dr. J. N. Frost was again in charge. The members of the Association were so interested in the operations that the meeting was not adjourned until six o'clock.

E. M. Pickens, *Secretary-Treasurer*

MONTANA VETERINARY MEDICAL ASSOCIATION

The twenty-second annual meeting of the Montana Veterinary Medical Associatlon was held at Great Falls, July 25-26, 1930. The Association met for its sessions at the new small-animal hospital of Dr. B. O. Fisher. About thirty veterinarians were in attendance, which is more than half the total veterinary population of the State.

The opening session was addressed by Mr. J. W. Freeman, president of the Great Falls Chamber of Commerce; Dr. W. J. Butler, State Veterinarian and the president of the Association, and Dr. G. E. Thomas, of Billings. The remainder of the morning session was devoted to routine business.

In the afternoon of the first day, Dr. E. D. Hitchcock, of the Great Falls Clinic, gave a very interesting and instructive talk on the development of biological products, such as the various gland extracts, and their use in medicine. Mr. H. B. Mitchell, former mayor of Great Falls and owner of a large dairy, spoke on certain observations made in the course of a trip through several European countries. Dr. T. F. Walker, health officer of Great Falls, was on the program but was unable to be present on account of sickness.

At the evening session, the Committee on Diseases gave its report, following which there was given opportunity for general discussion of any problem which any member might wish to bring up, with the chairman of the Committee on Diseases, Dr. Howard Welch, presiding.

In the morning of the second day, Dr. W. J. Butler spoke on the present status of infectious abortion control. This talk was followed by a general discussion, which was entered into by the owner of one of the large Great Falls dairies. This was followed by a small-animal clinic in Dr. Fisher's hospital, where several operations were performed. Methods of restraint were demonstrated and modern methods of diagnosis, including X-ray examinations of fractures and lung conditions.

In the afternoon, Dr. E. A. Tunnicliff, of the Veterinary Research Laboratory at Bozeman, gave an interesting discussion of the sheep and goat industry of West Texas, with special reference to disease control. Dr. A. F. Baldwin, of Miles City, told of finding fluke infestation in mink. This was of interest in view of the fact that larval stages of flukes have been found in trout in a Montana river, and it is possible that mink may be the hosts of the adults of this fluke. Dr. G. H. Ehlers, of Scobey, read a paper on the treatment of badgers infested with spirurid worms. He was successful in treating this condition with carbon bisulphid.

At the final business session, Bozeman was chosen for the meeting place in 1931. The officers elected were Dr. C. A. Hatterscheid, of Glendive, president; Dr. H. F. Wilkins, of Lewistown, vice-president; and Dr. H. Marsh, of Bozeman, secretary.

H. MARSH, *Secretary.*

CENTRAL CANADA VETERINARY ASSOCIATION

The midsummer meeting of the Central Canada Veterinary Association was held at the Animal Diseases Research Institute, Hull, Canada, on Aug. 30, 1930. Approximately sixty members attended each session and these were rewarded by one of the finest programs ever presented in Ottawa.

In the morning a conference on tuberculosis was held, at which the following papers were given:

"The Standardization of Tuberculin," by Dr. Geo. E. Stanley.
"Interpretation of Tuberculin Reactions," by Dr. J. McL. Stuart.
"No-Visible-Lesion Cases," by Dr. Orlan Hall.
"Paratuberculosisor Johne's Diease and the Johns in Test," by Dr. C. W. McIntosh.
"Bovine Subcutaneous Tuberculosis and the Relationship of Tuberculosis of Cattle to Tuberculosis of Other Animals and Chickens," by Dr. Chas. A. Mitchell.

This conference was followed by a discussion of the problem of tuberculosis, resulting in an exchange of views on different phases of this question. A conference on infectious abortion was also held, at which the following papers and demonstrations were presented.

"A Few Facts about Infectious Abortion upon which Control Measures are Based," by Dr. Chas. A. Mitchell.
"The Relation of the Practitioner to Infectious Abortion Control Work," by Dr. Orlan Hall.
"A Visit to the Serological Laboratory and Demonstration of the Various Blood Tests," by Dr. F. A. Humphreys.

This conference was supplemented by a most interesting paper which was given in the evening, on "Abortion Experiments on Guinea Pigs," presented by Dr. Ronald Gwatkin, of the Ontario Research Foundation of Toronto. In this interesting paper were given the results of experiments with different preparations of *Brucella abortus* organisms killed in various manners. Dr. Gwatkin showed that none of these dead antigens had any protective value against infection. The various papers on the subject of infectious abortion resulted in a mutual exchange of a great deal of useful information to all concerned.

In the afternoon and also in the evening was held one of the largest and most interesting clinics ever presented before the Association. The surgeons were Dr. W. J. R. Fowler, Professor of Surgery at the Ontario Veterinary College; Dr. A. A. Etienne, of Montreal, and Dr. David Caley, of Orillia. Fourteen major operations were performed upon large animals for the relief of such conditions as cryptochridism, poll-evil, fistulous withers, strangulated hernia, new growths, etc. Many small animals

were presented for operation and not until almost one o'clock in the morning did the clinic end. The wealth of clinical material gave the surgeons an opportunity to demonstrate some very useful operations that cannot fail benefiting the practitioner.

The amphitheatre at the Institute, which was lighted by a hospital operating-room system and is equipped with sterilizing appliances, running water, etc., made an incomparably fine place to carry out major operations.

During recess, between afternoon and evening sessions, a complimentary dinner was held in honor of the visiting surgeons, in the historic Standish Hall, which is situated on the ground over which Champlain trod on his way to the Upper Ottawa. Following the dinner the president of the Association, Dr. C. M. Higginson, took the opportunity of conveying to the visiting surgeons the thanks of the Association for their kindness in assisting in the program.

It was generally agreed by those present that the clinic was one of the finest ever witnessed, not only when compared with clinics held by small associations but also when compared with clinics held by the larger organizations. The spirit of goodwill which was so apparent also added to the pleasure of this meeting.

CHAS. A. MITCHELL, *Secretary-Treasurer*

BAY COUNTIES VETERINARY MEDICAL ASSOCIATION

Members of the Bay Counties Veterinary Medical Association were exceedingly fortunate to have the following members of the veterinary profession as guest-speakers at a dinner meeting held Tuesday evening, September 2, 1930, at the Elks' Club, San Francisco, California:

Dr. H. Preston Hoskins, Secretary-Editor of the American Veterinary Medical Association, Detroit, Mich.; Dr. J. C. Flynn, Kansas City, Mo.; Dr. C. H. Cocault, Iowa State College, Ames, Iowa; Dr. A. R. Glaisyer, Kauai, Hawaii; Dr. D. W. Hurst, Tecumhse, Nebr.; Dr. H. J. Stafseth, Michigan State College, East Lansing, Mich.; Dr. G. G. Graham, president of the Jensen-Salsbery Laboratories, Inc., Kansas City, Mo.

An appropriate program had been arranged for the occasion, including a brief report of the Los Angeles convention of the

A. V. M. A. by Dr. C. M. Haring, of the University of California, and a report on "Small Animal Hospitals in Los Angeles and Their Methods," by Dr. John McInnes, of San Francisco.

(Signed) G. M. SIMMONS, *Secretary.*

NORTHWESTERN ILLINOIS VETERINARY MEDICAL ASSOCIATION

The twelfth annual meeting of the Northwestern Illinois Veterinary Medical Association was held at the Court House, Freeport, Ill., October 1, 1930. About thirty veterinarians were in attendance. Problems in connection with the control of bovine tuberculosis occupied the afternoon session and abortion disease of cattle the evening session. Officers for the year were elected as follows: President, Dr. G. H. Norris, Pecatonica; vice-president, Dr. H. C. Curtis, Polo; secretary-treasurer, Dr. Roy E. Kluck, Freeport.

Alabama Now Has Tuberculosis-Free County

Systematic testing for the eradication of bovine tuberculosis on the area basis in Alabama has resulted in federal recognition of Montgomery County as the first modified accredited area to be established in the State. The accreditation was announced September 1. Alabama is the thirty-ninth state to have one or more counties so recognized by the U. S. Department of Agriculture. The prominence of Montgomery County as one of the best live stock counties of the South adds to the importance of the event.

An outstanding feature in the testing of the 41,207 cattle in the county was the finding of only two reactors. This remarkably small number is attributed by Dr. C. A. Cary, State Veterinarian, to enforcement of a state law which requires that all incoming cattle be tuberculin-tested and thus excludes all tuberculous animals. Alabama was one of the first states to establish such a requirement.

Another factor which influenced the health of cattle in Montgomery County, Doctor Cary explains, is that, in compliance with city health ordinances, tuberculin-testing has been carried on under the accredited-herd plan for the last ten years. Alabama was one of the first states to require the prompt slaughter of all cattle reacting to the tuberculin test, an important point in preventing the spread of tuberculosis among cattle.

NECROLOGY



ARTHUR HOWELL GRIFFITHS

Dr. Arthur H. Griffiths, of Montrose, Colorado, died at his home, June 4, 1930, as a result of complications developing from an attack of measles.

Dr. Griffiths was a graduate of the Colorado Agricultural College, class of 1912. He had been in practice at Montrose ever since graduation and also had considerable farming interests there. He was municipal food inspector for Montrose and also veterinarian to the State Penitentiary and State Reformatory farms. He is survived by his widow and four children. He was a member of Alpha Psi Fraternity.

JOHN W. ARNOLD

Dr. John W. Arnold, of Riverside, California, died suddenly August 15, 1930. Although he had been sick for over a year, hope had been held out for his recovery.

Born in Sandwich, Ill., Dr. Arnold attended local schools and the Bryant Business College, of Chicago, and then entered the McKillip Veterinary College. He was graduated in 1908 and almost immediately located at Riverside, Calif.

Dr. Arnold joined the A. V. M. A. in 1910. He was a member of the California State Veterinary Medical Association, an Odd Fellow and an Elk. He is survived by his widow.

JOSEPH H. CALDWELL

Dr. Joseph H. Caldwell, of Coutts, Alberta, passed away at his desk, August 25, 1930. Although he had not been in the best of health for several years, Dr. Caldwell had been able to look after his official duties as an inspector in the Health of Animals Branch, Canadian Department of Agriculture, a position which he held practically from the time of his graduation from the Ontario Veterinary College, in 1907. Most of this time has been spent in

the province of Alberta where he was found a very capable and efficient veterinarian at all times.

Dr. Caldwell joined the A. V. M. A. in 1911. He is survived by four daughters. Interment was at Carp, Ontario.

J. C. H.

ELLERTON ARTHUR RICHARDSON

Dr. Ellerton A. Richardson, of Glencross, South Dakota, committed suicide by slashing his throat with a knife, August 28, 1930. He had been in ill health for some time and this is believed to have brought on despondency.

Born in Paisley, Ontario, April 18, 1873, Dr. Richardson attended local schools and entered the Ontario Veterinary College while in his early teens. Although he finished the course earlier, he was not granted his diploma until 1892. He practiced with his father, Dr. J. J. Richardson (Ont. '71), at Paisley, for two years, and then went to Manitoba, where he practiced with the late Dr. J. G. Rutherford for two years. He then came to the United States and located at Anderson, Indiana. After practicing there for several years, he removed to Waterville, Minn., where he remained for four years.

Later Dr. Richardson went to Saint Paul, Minn., where he was veterinarian to the firm of Barrett and Zimmerman, horse dealers, at the Minnesota Transfer. Shortly afterwards he removed to Marcus, Iowa, where he remained until the Boer War broke out, when he went to New Orleans, to look after the shipment of horses purchased by the British Government for use in South Africa. He accompanied one shipment and remained in South Africa for four years. Upon his return to this country, Dr Richardson located at Goldfield, Iowa. Eight years ago he bought a small farm and ranch in the western part of South Dakota, near Glencross. He gave up practice, at this time, as his health was failing.

G. P. McC.

THOMAS FALCONER

Dr. Thomas Falconer, of Alexandria, Minn., died at St. Lukes Hospital, Alexandria, September 2, 1930, as the result of a fractured skull received in an automobile accident the day before.

Born July 16, 1858, at Lucknow, Ontario, Dr. Falconer came to the United States at the age of nineteen. He was graduated

from the Ontario Veterinary College in 1894. He first practiced in North Dakota and then located at Glenwood, Minn., going to Alexandria about 1901.

Dr. Falconer joined the A. V. M. A. in 1903. He was a member of the Minnesota State Veterinary Medical Association. He is survived by his widow (née Mary Douglas), one son and one daughter.

CHARLES J. SIHLER

Dr. Charles J. Sihler, veterinarian for the Kansas State Board of Agriculture, pioneer manufacturer of anti-hog cholera serum, first inspector in charge of federal meat Inspection at Kansas City, and one of the most prominent veterinarians of the Middle West, died at Kansas City, September 27, 1930. He had been ill for some time and became worse about a month before his death.

A native of Canada, Dr. Sihler secured his veterinary education at the Ontario Veterinary College. After his graduation, in 1893, he came to the United States and entered the service of the recently created Bureau of Animal Industry. He was assigned to Kansas City, Kansas, and placed in charge of meat inspection there.

Dr. Sihler was identified with the early history of the Kansas City Veterinary College. He taught meat inspection, as well as allied subjects, and at one time served as president of the College, which conferred on him the degree D. V. S., in 1901.

Dr. Sihler is usually credited with having established the first plant for the commercial production of anti-hog cholera serum. The Sihler Serum Company was established in 1909, and at the time of his death Dr. Sihler was president and treasurer of the Company. He always took an active interest in professional affairs, and was also favorably known in his own city, where he took a deep interest in civic matters.

Dr. Sihler joined the A. V. M. A. in 1893. He is survived by his widow, one son and one daughter. His father is still living in Ontario and there are several brothers and sisters also surviving.

C. C. D.

CLIVE W. MOOBERRY

Dr. Clive W. Mooberry, of Wauwatosa, Wis., ended his life by shooting himself, October 15, 1930. He had been in ill health for the past few months.

The deceased was a graduate of the Chicago Veterinary College, class of 1911, and practiced at Morton, Ill., for a number of years. He was president of the Lone Pine Fox Farm, at Random Lake, Wis., and he was financially interested in fox farms at Manitowoc and Kewaunee.

Dr. Mooberry joined the A. V. M. A. in 1912. He is survived by his widow and one daughter.

EDWARD GRAHAM FOLSOM, JR.

Dr. Edward G. Folsom, Jr., of Detroit, Mich., died suddenly the night of October 17, 1930, as he was preparing to leave his office on a professional call. He had been a sufferer from heart trouble for several years.

Born April 8, 1886, at Mount Clemens, Mich., he attended local grade and high schools, entering the Ontario Veterinary College in 1906. He was graduated in 1908 and later returned to his Alma Mater for another year of study (1919-20). Prior to coming to Detroit, Dr. Folsom was located at Fairmont, W. Va., where he was chief veterinarian for the Consolidated Coal Company. He was assistant to the late Dr. S. Brenton, of Detroit, at the time of the latter's death, five years ago, and succeeded to the large general practice which Dr. Brenton had established.

Dr. Folsom joined the A. V. M. A. in 1915. He was a member of the Southeastern Michigan Veterinary Medical Association.

His mother, two brothers and a sister survive him. His father, the late Dr. Edward G. Folsom, died about two years ago. He was health officer for Mount Clemens for many years and the Amercan Legion post there is planning a memorial for him.

DENNIS B. FITZPATRICK

Dr. Dennis B. Fitzpatrick, of Falfurrias, Texas, died at his home, October 19, 1930.

Born in Philadelphia, Pa., January 3, 1866, Dr. Fitzpatrick attended parochial schools, LaSalle College and the University of Pennsylvania. He was graduated from the latter institution in 1893 and entered general practice in Philadelphia, where he remained for over twenty years.

During the late war Dr. Fitzpatrick enlisted in the Veterinary Corps. After the war he went to Texas and resumed general practice, at Falfurrias.

Dr. Fitzpatrick joined the A. V. M. A. in 1908. He was a member of the State Veterinary Medical Association of Texas. He was a fourth degree member of the Knights of Columbus and was Grand Knight of the Falfurrias Council at the time of his death. He was also a member of the American Legion and the Elks Lodge No. 2 of Philadelphia. He is survived by one daughter and one sister.

Our sympathy goes out to Dr. Harry Morris, of Baton Rouge, La., in the death of his wife, September 15, 1930, at New Orleans, following an emergency operation for appendicitis on June 20; and to Dr. L. B. Michael, of Collinsville, Ill., and Dr. Wm. R. Michael, of Highland, Ill., in the death of their father, in his 84th year, October 21, 1920.

PERSONALS

MARRIAGES

Dr. J. L. Johnson (K. C. V. C. '11) to Miss Myrtle Tipton, of Sylvester, Ga., August 26, 1930.

Dr. R. J. Potteiger (U. P. '18) to Miss Marian Moyer, both of Selinsgrove, Pa., August 30, 1930.

Dr. A. L. Stigers (Iowa '30), of Williamsburg, Iowa, to Miss Marie Galsen, of Woodbine, Iowa, August 10, 1930.

Dr. W. H. Dunham (McK. '15), of Delavan, Wis., to Miss Leona Green, of Morrison, Ill., October 4, 1930, at Morrison, Ill.

Dr. Murrell O. Robinson (U. P. '25), of Philadelphia, Pa., to Miss Elizabeth Virginia McKibbin, October 1, 1930, at Waynesboro, Pa.

PERSONALS

Dr. J. P. Miller (St. Jos. '20) has resumed general practice at Boonville, Mo.

Dr. Clyde Dohner (Ont. '28) has located at Reedsburg, Wis., for general practice.

Dr. Norman H. Brinkman (Iowa '25), formerly of Collins, Iowa, gives a new address: Colo, Iowa.

Dr. William H. Dunn (U. P. '30) has located at Media, Pa., where he is engaged in general practice.

Dr. John K. Morrow (O. S. U. '30) is now associated with Dr. M. L. Morris (Corn. '26), at Stelton, N. J.

Dr. W. E. Neary (Colo. '21) has requested a change of address from Moscow, Idaho, to Box 809, Boise, Idaho.

Dr. Charles R. Schroeder (Wash. '29) has accepted a position at the Lederle Laboratories, Pearl River, N. Y.

Dr. Fay G. Rankin (Colo. '28), formerly of Astoria, Ore., is now located at Saint Helens, Ore. Address: Box 704.

Dr. E. K. Glover (K. C. V. C. '12), formerly of Kansas City, Mo., is now located at 513 E. Losey, Galesburg, Ill.

Dr. T. E. Nichols (O. S. U. '21), formerly of Columbus, Ohio, gives his new address as 5041 W. Broad St., Camp Chase, Ohio.

Dr. J. J. Thomas (U. P. '17), formerly located at Etna, Pa., gives his new address as 112 Highland Ave., Clarks Summit, Pa.

Dr. Haim Applebaum (U. P. '28) is with the Palestine Army with the title of V. F. D. O., and was recently located at Shamkh.

Dr. B. B. White (K. S. A. C. '20), has removed from San Francisco to Berkeley, Calif. His new address is 1204 Delaware St.

Dr. W. E. Sailor (Chi. '12), of Stronghurst, Ill., was kicked by one of his equine patients, recently, and sustained fractures of three ribs.

Dr. Lyman R. Vawter (K. S. A. C. '18) is on a year's leave of absence from the University of Nevada, and is spending the year at Cornell University.

Dr. O. W. Snyder (Amer. '84) and Dr. Charles H. Gates (Ind. '21), of Liberty, Ind., have dissolved their partnership. Both will remain in Liberty.

Dr. W. N. Armstrong (Ont. '94), of Concord, Mich., was reelected County Veterinarian by the Jackson County Board of Supervisors, at the October meeting.

Dr. E. J. Watters (Chi. '07), of Calumet, Mich., was recently appointed official veterinarian for the village, in connection with the local meat inspection ordinance.

Dr. C. D. McGilvray (Ont. '00-McK.'01), principal of the Ontario Veterinary College, Guelph, Ontario, was a visitor to Manitoba during the month of September.

Dr. Jean Smith (Corn. '29) recently accepted a position with the Borden Milk Company, and has been assigned to territory in the northwestern part of Pennsylvania.

Dr. M. W. Shempf (McK. '02), who recently disposed of his practice at Tuscola, Ill., has removed to Normal, Ill., where he plans to conduct a hospital for small animals.

Dr. Aubrey M. Lee (K. S. A. C. '22), who spent the past year in postgraduate work at Ohio State University, has returned to the University of Wyoming, at Laramie.

Dr. Jacob Z. Gehres (U. P. '21), formerly of Reading, Pa., has purchased the practice of Dr. T. F. Moran (R. C. V. S. Dublin '19), at Pittsburgh, Pa. Dr. Moran has returned to Ireland.

Dr. Fred C. Schmidt (Ont. '21) and Dr. Wm. E. Ruggles (Colo. '27) recently announced the opening of their new small-animal hospital, located at 14th St. and Lovejoy Ramp, Portland, Ore.

Dr. B. F. Humphreys (McGill '00) recently announced the removal of his veterinary hospital from 939 E. 42nd Place to 824 E. 43rd St., between Drexel Blvd. and Cottage Grove Ave., Chicago.

Dr. H. C. H. Kernkamp (O. S. U. '14), of the University of Minnesota, and Dr. D. I. Remington (K. C. V. C. '13), of Tracy, Minn., were on a pheasant hunt together, about the middle of October.

Dr. A. H. Kettelhon (McK. '15), of Columbus, Wis., lost his veterinary hospital in a serious fire which, for a time, threatened the destruction of all buildings in the same block, about the middle of September.

Dr. John A. Schaefer (Gr. Rap. '13), of Bangor, Mich., has been appointed a member of the Michigan State Board of Veterinary Medical Examiners, succeeding Dr. Burton A. Perry (Gr. Rap. '12), of Hastings, resigned.

Dr. William C. Nickel (U. P. '16), of Nazareth, Pa., has been selected to act as county chairman of the Democratic Committee in Northampton County. He recently sponsored a rally which was the largest in the history of that County.

Dr. Reuben Hilty (O. S. U. '07), of Toledo, Ohio, attended the meeting of the American Association of Zoological Parks and Aquariums, in Saint Louis, recently, and delivered an address, entitled, "The Value of Scientific Veterinary Service in a Zoological Park."

Dr. G. W. Butler (Ont. '84), of Indianapolis, Ind., was retired from the service of the U. S. Bureau of Animal Industry, the latter part of August, after thirty-one years of service, most of which had been in connection with meat inspection and stock yards inspection.

Dr. C. E. Turnbull (Gr. Rap. '13), who has been associated with Dr. A. L. Tow (Ind. '18), in general practice in Detroit, Mich., for several years, has withdrawn from the partnership and gone into practice on his own account at 13121 Michigan Ave., Dearborn, Mich.

Dr. Bloomer E. Myers (Ind. '13), who practiced in Plainville, Ind., for about fifteen years, has removed to Washington, Ind., and opened an office there. Dr. Myers will devote a part of his time to tuberculin testing for the State, in Steele and Washington townships.

Dr. E. C. McCulloch (K. S. A. C. '24), who has been at the Alabama Polytechnic Institute, Auburn, the past year, has accepted an industrial fellowship offered by the Pennsylvania Salt Company, and is now working in the Department of Veterinary Science, at the University of Wisconsin, Madison.

Dr. E. C. W. Schubel (U. S. C. V. S. '11), of Blissfield, Mich., attended the Short Course in Diseases of Poultry, at Ohio State University, in October, and had a serious automobile accident on the way home. About seven miles from Blissfield, Dr. Schubel ran into a double team of horses, killing one outright. The automobile was completely wrecked, but Dr. Schubel escaped injury.

Dr. John Patterson (Iowa '12), who has conducted a general practice at Hedrick, Iowa, for over eighteen years, has decided to give up long nights and muddy roads and has gone to Manhattan, Kans., where he will be associated with Dr. N. D. Harwood (K. S. A. C. '18), under the name of Patterson-Harwood Laboratories. Dr. Patterson is president and Dr. Harwood is secretary-treasurer of the concern, which will engage in the manufacture and distribution of veterinary biologics. Dr. Patterson considers that Manhattan offers wonderful facilities for educating his children.